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# FURTHER OBSERVATIONS OF MASTOID STRUCTURE BY MEANS OF THE X-RAY.*

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## PREFACE.

Several years ago we undertook some X-ray investigations of the mastoid, the results of which were published in two papers.[1] [2] In these, we attempted to correlate the anatomic data obtainable by mastoid radiography with the clinical and surgical aspects of mastoid disease. As these preliminary studies were limited to clinically observed cases, it became evident that certain anatomic features could be more accurately determined and perhaps standardized by X-raying a large number of bones. In casting about for material, we naturally turned to Dr. B. Alex. Randall, whose collection of temporal bones is world famous. On requesting the privilege of radio-

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graphing these bones, we learned that Drs. Joseph C. Beck and Samuel Iglauer had previously made a similar request. At the suggestion of Dr. Randall, it was decided that we all join forces, we ourselves undertaking the mechanical work of making the radiograms. This we have been doing for the past two years, but as the work is more time consuming than first anticipated it has made slow progress and even now is in nowise completed. While experimenting with various angles for projecting the mastoid on to the X-ray plate, we discovered an angle that revealed the cellular structure of the mastoid proper as did no other. This, which we have termed the occipital projection, is equally applicable to the living and now constitutes a part of our routine examination, being especially useful where the cell structure is limited as is the case in infants and young children. We have also found this projection most useful in correlating X-ray appearances with the modern ideas of mastoid development and structural variations so carefully worked out by Wittmaack.[3] As our work along this line is still incomplete, it was deemed advisable to present the new projection together with its practical applications as a preliminary report, this with the hearty assent of Drs. Randall, Beck and Iglauer.

It seemed desirable, however, to divide this into two parts, the one discussing the more strictly X-ray aspects while the other deals with anatomic and clinical correlations. Hence, those desirous of purely technical details are referred to the first part, which will shortly be published in the Journal of Radiology.

It might be well at this point to state our position as regards mastoid radiography in general. As brought out in the first part, we do not consider this method of examination as a means by which the otologist can shift the responsibility of making diagnoses or deciding for or against operation, and we believe that the radiologist who attempts to assume these responsibilities is in a fair way to descredit all mastoid radiography. We do know, however, that this is the only possible means we have of informing ourselves as to the anatomic structure of any given mastoid, unless we resort to mere guesswork or destructive exploration. As to the proof of this

statement, the hundred temporal bones so far examined were carefully studied externally and an estimate made as to internal structure before their subjection to X-rays. With all the advantage of no obtruding soft tissues coupled with exact knowledge as to the location of the sinus and the intracranial configuration, we were absolutely wrong in about 50 per cent of all cases. Certain types of infection in the living, to be sure, are coupled with certain anatomic peculiarities; but as to determining or even estimating the internal structure of a mastoid from its mere configuration, the above investigation speaks for itself.

Before proceeding with the main discussion, we wish to pay tribute to the kindness and courtesy of Dr. Randall, whose hearty cooperation has made this work possible.

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Although it is well known that the structure of one mastoid may be totally unlike that of another, yet this fact receives little or no consideration in everyday practice. In the clinical study of a middle ear infection with possible mastoid involvement it is usually assumed, perhaps unconsciously, that the mastoid contains a certain amount of pneumatic cellular structure. That there is one chance in five that this is not the case was brought out by Cheatle,[4] [5] some fifteen years ago, when he demonstrated that 20 per cent of all adult mastoids contain practically no pneumatic cells, the "mastoid mass" remaining diploetic or dense as in infancy. These figures were recently confirmed by Turner[6] and Porter, so far as European skulls are concerned. They also made the interesting observations that the percentage of infantile mastoids was greater in dolicocephalic skulls than in those of the brachycephalic type and that infantile mastoids were decidedly less prevalent in primitive races than in those on a higher plane of civilization. As the cell content of the individual mastoid ranges from none at all up to the most widespread distribution, these variations in structure apparently signify different degrees of development, with the infantile types at one extreme and the completely pneumatic at the other, while the numerous varieties of mixed mastoids represent the intervening stages.

It seems self evident that the extent of an acute mastoid infection together with its clinical course, in fact, its entire

pathology, must depend in large measure upon the amount and the distribution of the pneumatic cells. While there is some dispute as to how much reliance should be placed in X-ray findings as regards pathology, this is our sole means of information both as to the amount and the distribution of the pneumatic cells, short of mere guesswork or destructive exploration; though, judging by the X-ray reports as quoted in the current literature, this part of the examination is too often neglected. These reports deal with the question of haziness, cloudiness or the breaking down of cell walls and the like, but a report that gives the reader any definite idea as to the anatomic structure of the mastoid in question is decidedly rare. Just why important anatomic details that can be easily determined should receive so little attention is hard to fathom, but is probably due to inadequate and unstandardized methods for describing the same. As the usual classification of mastoids into three types, diploetic, pneumatic and mixed, is altogether too general for either description or comparison, a more precise classification would seem highly desirable. Several such have been proposed, one of which was devised by ourselves.[1] The latter is not purely arbitrary, but is based on the development of the mastoid, the various types described representing different degrees of development. It would seem as though such a classification ought to prove useful in describing and comparing the various types of mastoids encountered in everyday practice. Nevertheless, this classification, though perhaps possessing some merit, has proven, like all others, to be utterly useless for the purpose intended. This is due to the fact that the various types of structure are not so sharply differentiated as an abstract description would seem to indicate, but as actually encountered one type shades into another so gradually that clear cut divisions are impossible. Therefore, the only other alternative is to devise some practical method of accurately portraying and describing the anatomy of each individual mastoid in terms that are intelligible to the average reader.

The recent work of Wittmaack,[3] "The Normal and the Pathological Pneumatization of the Temporal Bone," supplies a terminology that is both comprehensive and easily understood, while the occipital X-ray projection, which discloses

all pneumatic cell structure, renders practically every mastoid amenable to concise and accurate anatomic description. Wittmaack has demonstrated that by the end of the first year the pneumatic structure of the mastoid consists of a large antrum with comparatively smooth walls. Under normal conditions pneumatic cells then began to develop in two directions: concentrically into the lumen of the antrum and excentrically toward the periphery. In concentric pneumatization, bony trabeculæ sprout from the walls of the antrum underneath the mucous membrane. By their fusion one with another these trabeculæ form first one layer of the fine pneumatic cells on the walls of the antrum and then others till the antral lumen becomes much narrowed. In excentric or peripheral pneumatization the original marrow spaces or diploe are replaced by pneumatic cells, all of which is described with the utmost histologic detail. As pneumatization progresses toward the periphery the cells become larger and larger, finally ending in a series of broad cul de sacs. These large peripheral cells are also partially subdivided by bony trabeculæ that sprout up as in the antrum. This is a fact that should always be borne in mind when attempting to diagnose a pathologic breaking down of cell walls, for this appearance may be due to a normal but incomplete subdivision of the larger cells. In fact, such areas showing an apparent breaking down of cell walls are fairly common in the peripheral cells of absolutely normal mastoids. (Fig. 1, 2.) Radiograms of bones are used to illustrate this point because they can be reproduced much better than those from the living. However, the same appearance is frequently seen in the latter. If there be a certain amount of haziness in such cases, either from pathology or poor technic, there is quite likely to be some discrepancy between the X-ray reports and the operative findings, which probably accounts for some of the belief that mastoid radiograms are quite apt to be misleading.

According to Wittmaack, a mastoid that is not completely pneumatized by the end of the third year or, at the very latest, by the end of the fifth, never becomes so later on. Furthermore, in a completely pneumatic mastoid, the process of pneumatization never comes to a complete halt but continues on into old age, though at a decidedly slower pace.

In arrested development the disturbing factors—a detailed description of which is beyond the scope of this paper—have played their part by the end of the first year, and the earlier in life they come into play the more profound will be their effect on the process of pneumatization. In other words, restricted pneumatization is not due to infections that occur in later life. The following case has considerable bearing on this point: Figure 3 is a radiogram of Case 10 from our report of several years ago.[1] This was taken three and a half years after an acute mastoid infection that recovered without operation, though with some residual deafness and subjective symptoms. There is considerable density posteriorly, in the midst of which are scattered scraps of pneumatic cells, evidently sclerosis due to infection in adult life, as is seen when compared with the normal mastoid. (Figure 4.) Figure 5 is a radiogram of the same mastoid taken in November, 1922, or eight and one-half years after the original infection, and five years after the plate shown in Figure 3. While it may not be so evident in the reproductions, yet a comparison of the original radiograms shows a decided increase in the pneumatic cellular structure; whereas, according to our old conceptions, a decrease or at least a standstill is to be expected.

Wittmaack describes the completely pneumatic mastoid as follows: "In accordance with the somewhat different procedure in development, the cell net surrounding the antrum is relatively fine meshed, while the meshes gradually widen out toward the periphery and finally end in a fringe of large bladderlike cells. The partitions of this network consist of comparatively thin bone of about the same thickness." This conception of pneumatic structure as a cell net constitutes a reliable basis for describing the anatomy of all mastoids, irrespective of age or type of structure, providing all of the cell net be projected on to the X-ray plate and none of it be hidden by the petrous pyramid. In plates taken in the usual projection with the two meati superimposed, the pyramid extends backward and obscures the cell net in the vicinity of the antrum. (Fig. 6a.) While this may be of no great moment in large pneumatic mastoids, it is of the utmost importance in the examination of young children or those cases in which the extent of the cell net is decidedly limited. In the occipital

projection the pyramid is thrown forward and overlies the middle ear and the zygoma (Fig. 6b). As this obscures the zygomatic cells, both projections are necessary for a complete examination.

The foregoing description of the normal pneumatic cell net coupled with the occipital projection elucidates a point that has long defied our attempts at explanation, namely, the apparent presence of diploe about the antrum of otherwise completely pneumatic mastoids. Other observers have likewise been puzzled by this appearance, which has been described as a "dumbbell constriction in and about the antrum." Bearing in mind Wittmaack's description, it will be seen on comparing Figures 6a and 6b that this diploetic appearance is due to the fine meshed cell net, that normally surrounds the antrum, showing through the superimposed pyramid. These fine meshes of the normal cell net are apt to be mistaken for diploe, even in sectioned bones, as shown by Figure 6c, which is the same bone as in Figures 6a and 6b. On comparing these three figures, it will be seen that the occipital projection roughly corresponds to a vertical cross section of the mastoid in the sagittal planes.

The mastoid with no pneumatic cells presents the greatest contrast to the completely pneumatic type, as shown by Figure 7a. This might be termed either a diploetic, an infantile, an acellular or a sclerosed mastoid or one with total arrest of pneumatization; but if the radiologist simply state that the entire mastoid is diploetic with no visible evidence of pneumatic cells, he will give us a clearer concept of its actual structure than if he merely give it a name. From this information the otologist will be able to visualize the anatomic relations fully as well if not better than by exploratory operation. He will know that the pneumatic structure of such a mastoid consists of an antrum deeply placed in dense bone; that there may be only a very thin plate of bone between the antrum and either the middle or the posterior fossa or the sinus or all three combined; that, as even the most virulent infection in an antrum so situated is incapable of producing but few if any of the typical signs and symptoms of mastoiditis, though all the more likely to give rise to intracranial

complications, an operation will be urgently indicated on far less clinical evidence than almost any other type of mastoid. In other words, he should not allow a patient to die of meningitis or some other intracranial complication while waiting for typical signs and symptoms of mastoiditis to make their appearance. Even though delay be unattended by disaster, it should always be borne in mind that with this type of mastoid a middle ear discharge is quite likely to become chronic. Hence, in view of the constant annoyance, the loss of hearing and the ever present menace to life thereby entailed, a simple mastoidectomy is often indicated for the prevention of this complication, even though there be no other sign or symptom, save otorrhea, to justify its performance.

That the occipital projection (Fig. 7a) is a reliable guide as to the absence of cells is demonstrated by sectioning the bone (Fig. 7b). From the standpoint of development, it makes little or no practical difference whether the mastoid be dense or diploetic. In calling attention to the two infantile types, the dense and the diploetic, Cheatle[4, 5] emphasizes the fact that in the latter a layer of compact bone always separates the antrum from diploe. He offers no explanation as to the occurrence of these types beyond suggesting an increased density of bone as a determining factor. From Wittmaack's description[3] of pneumatization it becomes evident that the layer of compact bone separating the antrum from diploetic structure is due to a conversion of the narrow spaces or diploe adjacent to the antrum into compact bone; while, in the case of the dense mastoid, this process is so exaggerated that the entire mastoid becomes sclerosed. In other words, these two types, the diploetic and the dense, are sclerosed mastoids due to total arrest of pneumatization, differing only in the degree of sclerosis. It seems evident, however, that osteomyelitis would be more probable in a diploetic than in a dense mastoid, especially if there be a small amount of pneumatic structure by which an antrum infection could be carried deeper into the mastoid. Wittmaack has further demonstrated that the bone itself plays but a secondary role in the process of pneumatization. If the bone be dense the cells may be smaller and the cell net not so extensive, but in this case, if other conditions be normal, the entire mastoid will be small yet perfectly pneumatized. The

occurrence of perilabyrinthine cells is added evidence that the density of bone per se is no bar to pneumatization, as the densest bone in the body is to be found in this region.

A very small fine meshed cell net is depicted in Figure 8a. This radiogram demonstrates the value of projecting the pyramid forward so as not to obscure the region of the antrum, as may be seen on comparing this radiogram with that shown in Figure 8b, taken in the usual angle with the two meati superimposed. That the occipital projection discloses practically all of the pneumatic cells that are present is again demonstrated by sectioning the bone (Figure 8c). This is of the greatest importance wherever the cell net is much restricted, as is usually the case in very young children. The superiority of the occipital projection at this age becomes apparent on comparing the mastoid radiograms of a twenty-two months old child (Figure 9a, 9b), taken with the usual and the occipital angles respectively. Whether or not this be brought out in the reproductions, a study of the original films discloses in the occipital projection a small but well marked cell net in the vicinity of the antrum, practically all of which is hidden by the pyramid when the two meati are superimposed. These two mastoids, the one in the dry bone and that in the child, can be accurately described as follows: X-ray examination discloses a much restricted cell net limited to the vicinity of the antrum, the remainder of the mastoid being entirely diploetic.

Infection in a mastoid of this type at this age or thereabouts will usually evoke the typical signs and symptoms of mastoiditis, though our experience has taught us not to rely on this too implicitly. In the case of the adult, however, we should always remember that even this grade of arrested development may be coupled with sclerosis sufficient to mask the typical signs and symptoms of mastoiditis almost as effectually as in the acellular types. Hence the slightly pneumatized mastoid is, potentially, the most dangerous of all; for the small amount of pneumatic structure predisposes to a deeper and, at the same time, to an insidious extension of virulent infection.

A more advanced stage of development is shown in Figure 10. The cell net is typical in that its meshes are small in the antral region and gradually widen out toward the periphery.

It is limited, however, to the upper two-thirds of the mastoid, the tip being entirely diploetic. This illustrates another important point brought out by Wittmaack in the course of his investigations, namely, that while the upper part of the mastoid is pneumatized by cells that push out from the antrum, the tip is pneumatized from the hypotympanum. The aditus serves as the outlet to the middle ear for the upper group of cells, while the sinus tympani, located in the posterior pole of the hypotympanum, performs this function for those in the tip of the mastoid. In the normal course of development one cell system so blends with the other that the difference in outlet becomes unimportant.

Complete pneumatization of the upper two-thirds of the mastoid with a total arrest at the tip is fairly common. The reverse of this condition, while possible, seems to be extremely rare, and we have found no reference in Wittmaack to any such type of structure. Be that as it may, within the past year we encountered at operation a case of this type in which, unfortunately, the indications for operation were too evident and too urgent to permit of an X-ray examination. The upper half of the mastoid was entirely dense, while the entire tip and the bone for an inch posteriorly contained large pneumatic cells filled with pus. A chain of small deeply placed cells extended upward along the posterior canal wall and ended abruptly at the approximate level of the hypotympanum, though a due respect for the facial nerve prevented an exact determination of this point. It might be interesting to note that this patient, a child of twelve, had been in extreme pain for two weeks with no aural discharge at any time or, for that matter, no paracentesis. When examined previous to operation the drum, though congested, showed no bulging. Though mastoids that are dense or diploetic in the antral region and pneumatic below seem to be most unusual, those whose antral cell net is much restricted while the tip is completely pneumatic are of comparatively frequent occurrence. A good example of this condition is shown in Figures 11a, 11b and 11c.

In a previous communication[1] we described a type of mastoid, long known to otologists of the Massachusetts Charitable Eye and Ear Infirmary as the "double decker." The cells of

a "double decked" mastoid are arranged in two tiers, an inner and an outer, which are separated by a layer of bone closely resembling the inner table. We attempted to explain this type as a mixed mastoid with diploe in one plane and pneumatic structure in the other, but in the light of Wittmaack's* observation that the mastoid is pneumatized not by one cell system alone but by two which, at first, are separate and distinct, it seems probable that the "double decked" arrangement is due to the antral cell system failing to blend with that from the hypotympanum. Occasionally the squama, which forms the outer antral wall and approximately the upper third of the mastoid cortex, extends downward over the petrosa nearly to the tip of the mastoid. It seems possible that this overdevelopment of the squama may be coupled with an increased resistance along the plane of the squamomastoid suture sufficient to explain a lack of fusion of the two cell systems. At all events, this type of mastoid generally has a few large cells at the tip, and at operation the "double decked" condition is usually unsuspected until these tip cells are uncapped. As the clinical significance and the X-ray examination of this type of structure have been fully discussed in the previous communication,[1] to deal with them here would be mere repetition. It seems well to state, however, that the radiologist who does not make stereoscopic study a part of his routine examination is not likely to forewarn the otologist as to this type of structure.

Again consulting Figure 10, it will be seen that in addition to the diploetic tip the cell net is somewhat restricted posteriorly, the cells in contact with diploe having thickened walls. According to Wittmaack, this latter condition is characteristic of arrested pneumatization due to hyperplasia of the lining mucosa. This increased density of bone at the periphery of the cell net, though probably serving as a barrier against the spread of infection by contact, has other accompaniments that render the intracranium more susceptible to blood stream infection. As an explanation of these points involves the entire question of mastoid development, it seems proper to

*Wittmaack does not mention the "double decked" type of mastoid.

digress for the moment in order to mention the salient histologic features of this process.

The fetal middle ear cavities—tympanum, recessus and antrum—are first blocked out by bony—that is to say, cartilaginous walls, and are filled with a gelatinous embryonic tissue known as mesenchyme. A lumen begins to develop between the fifth and sixth month of fetal life by an evagination of epithelium from the eustachian tube. As the lumen expands the embryonic tissue recedes so that the mucous membrane that lines these cavities is, at first, normally hyperplastic and consists of epithelium originating from the eustachian tube and subepithelial tissue derived from mesenchyme. During this time capillaries, channeling in from the jugular bulb, from the dura of both fossæ and from the knee of the lateral sinus, come into intimate contact with the submucosa of the developing pneumatic cavities and their respective cell systems. Under normal conditions, this hyperplastic mucosa regresses to a mucoperiosteal membrane of such thinness that individual layers can no longer be differentiated, the vascular communications with the dura, the sinus and the jugular bulb being thereby obliterated. In the tympanum, the recessus and the antrum this regression is either completed by the end of the first year or probably not at all; if the mucosa remain hyperplastic its vascular connections also persist. The completion of the middle ear cavities marks the end of the first period of development; the second, which is characterized by the formation of the pneumatic cells, closely follows.

Both the hypo- and epitympanic as well as the antral cells are formed in two ways: First, by the fusion of bony trabeculæ that sprout from the walls of the respective cavities, followed by evagination of the lining mucosa into the spaces thus formed; second, by progressive transformation of the adjoining marrow spaces into pneumatic cells. Before these spaces actually become pneumatic they undergo preliminary transformation. As the mucosa of the middle ear cavities has previously regressed to a thin mucoperiosteal membrane the mucosa adjacent to a marrow space, about to be pneumatized, becomes vascular and somewhat thicker. A cone of this thickened subepithelial tissue then forces its way into the marrow

space and displaces the marrow content. The continued growth of this cone causes an absorption of bone, with the result that several spaces are thus thrown into one. The perivascular tissue of capillaries from the dura of both fossæ, the lateral sinus and the jugular bulb, respectively, also take part in this preliminary process. Hence, when the former marrow space becomes pneumatic by evagination of epithelium from the adjoining pneumatic cavity the mucous membrane that lines the newly formed cell is at first normally hyperplastic and has the same vascular connections as the respective middle ear cavity from which it originated. These are, likewise, severed when the mucosa regresses to a thin mucoperiosteal membrane; but if regression does not occur and the mucosa remains hyperplastic these vascular communications with the dura, the sinus or the jugular bulb also persist. Moreover, instead of an absorption of bone, as at first, a deposition of bone then ensues, causing the cell walls to become much thickened. If, during the first year, when the mucosa of the middle ear cavities is normally hyperplastic, some disturbing influence prevents its complete regression or renders it even more hyperplastic, the mucosa may be so thick that evagination of epithelium into the spaces prepared for the same becomes impossible. As this hyperplasia is limited to the superficial layers of the mucous membrane it does not interfere with preliminary transformation of the marrow spaces but, on the contrary, actually favors this process, so instead of these spaces being further transformed into pneumatic cells they are eventually converted into dense bone. In the case of the diploetic mastoid this explains the layer of compact bone that separates the antrum from diploe. So far as their development is concerned there is no essential difference between this and the completely dense mastoid, except that in the latter type the sclerosing process has been more active.

At first glance it would seem as though a deposition of bone sufficient to obliterate the marrow spaces would also obliterate the vascular connections of a hyperplastic mucosa, but such is not likely to be the case, for when bone opposes a blood vessel the bone, not the vessel, succumbs. An aneurism, for example, will invariably erode any bone in the body that tends to obstruct its growth. Therefore, whenever the radiogram dis-

closes an infantile type of structure or a restricted cell net whose peripheral cells show thickened walls the possible vascular communications with adjacent intracranial structures should be kept in mind. Dry temporal bones with restricted pneumatic development frequently show numerous pin point openings at the knee of the sinus. Whether or not these openings represent direct vascular communications between the mastoid and the sinus in the living is of course open to question, though they are certainly suggestive. This point could probably be determined by injecting fresh material or possibly wet specimens.

As mentioned before, the tip of the mastoid is pneumatized not from the antrum but from the hypotympanum. In the preliminary transformation of the marrow spaces in this region the process begins at the sinus tympani and, proceeding backward and downward, takes in the marrow spaces surrounding the facial nerve. This is a point well worth remembering when extirpating the deeper cells of the posterior canal wall.

While certain disturbing influences* may affect the superficial layers of the mucosa with consequent persistence or even an exaggeration of the original hyperplastic condition, other causes may give rise to fibrosis of the deeper layers without superficial effects. In this case the submucosa will be inca-

*Wittmaack believes that hyperplasia of the mucosa is caused by the entrance into the tympanum of amniotic fluid, meconium, etc., during fetal life, or more probably at birth; or the entrance of vomitus, etc., during the first year of life; that this material sets up a foreign body irritation which is coupled with a low grade infection that is insufficient to produce symptoms such as perforation and discharge. Wittmaack believes that this accounts for the great frequency with which collections of pus are found at autopsy in the middle ears of young children. Solowzow found this condition in over 90 per cent of all children coming to autopsy. It has been designated otitis media concomitans, otitis media neonatorum and otitis media of the suckling. Fibrosis, on the other hand, he believes to be due to acute middle ear infection during the first year of life.

The recent investigations of Dean and his workers are most interesting. They have found collections of pus and a thickened mucosa in the sinuses and the middle ears of rats fed on a diet deficient in fat soluble vitamin A. This work is suggestive, at least, as regards otitis media neonatorum and the etiology of a hyperplastic mucosa.

pable of carrying out a preliminary transformation of the marrow spaces, while the epithelium may sink into them directly. In this event the pneumatic cells thus formed will be small and limited in extent (Fig. 12), though not to the same degree as in cases with intense hyperplasia. Such pneumatic cells will have neither the thickened walls nor the vascular connections that are found in arrested pneumatization with a hyperplastic mucosa. There are probably innumerable cases of arrested development in which both factors come into play.

This in barest outline is the process of normal and abnormal pneumatization, as described by Wittmaack, and is thrust into this particular paper because of its extreme importance in the interpretation of mastoid anatomy.

Another type of structure that requires mention is the overpneumatized mastoid. (Fig. 13a.) Wittmaack believes this is due to a hyperplasia of the mucous membrane too slight to prevent or even hinder evagination of the epithelium and yet sufficient to so stimulate preliminary transformation of the marrow spaces that not only the mastoid proper but the bone adjacent becomes widely pneumatized. The usual projection is more useful than the occipital for the study of this type of mastoid, as will be seen on comparing Figures 13a and 13b. The upper portion of the mastoid shown in Figures 14a and 14b is extensively pneumatized, while the tip is entirely dense. On the basis of Wittmaack's theories, this indicates that during the course of development there was a slight hyperplasia of the mucous membrane of the antrum sufficient to induce an overdevelopment of the antral cell system, while the mucosa of the hypotympanum was so intensely hyperplastic as to prevent pneumatization of the tip.

If the mucosa be somewhat more hyperplastic than was, presumably, the case in Figures 13a and 13b and the upper portion of Figures 14a and 14b, the cell net may be normal in extent, but with partitions between cells much broader and more diploetic than under normal conditions. This kind of structure is shown at the periphery of the cell net in Figures 15a and 15b. Though a mastoid of this type appear completely pneumatic, it may in reality be a mixed mastoid with

the pneumatic structure intermingled with diploe, in contradistinction to the other variety in which the two are segregated. (Figs, 8, 9, 10.)

Under these conditions it would seem quite possible for a mastoid infection to set up a low grade osteomyelitis in the framework of the cell net that might remain dormant for some time after typical signs and symptoms had disappeared, a condition that might account for some of the cases of socalled "latent mastoiditis." As it is obvious that no hard and fast line can be drawn between this and the completely pneumatic mastoid, the added information to be gained from stereo plates is usually necessary in order to distinguish between these two types.

The structure of the pneumatic mastoid permits of rapid and widespread infection, which often dies down about as quickly as it flares up. This behavior is usually attributed to the large size of the cells, which is presumed to insure good drainage—a most inadequate explanation at best—which in the light of present knowledge becomes absolutely untenable. For how can their mere size favor the drainage of the mastoid cells, inasmuch as they must all drain through the fine meshed cell net that surrounds the antrum?

As mentioned in our communication[1] of several years ago, changes in aeration that result from a middle ear and mastoid infection probably account both for the abrupt onset of stormy and threatening symptoms and for the rapid subsidence of the same, so often observed in the case of pneumatic mastoids. As an otitis media first renders the eustachian tube impermeable, absorption of air from the middle ear cavities and the pneumatic cells naturally follows, while the resulting venous congestion of the lining mucosa is the best possible preparation for a rapid spread of infection. It seems probable, however, that incision of the drum membrane or even spontaneous perforation not only promotes drainage but also tends to reaerify the middle ear and the mastoid cells, thereby restoring the air pressure within these cavities, approximately, to that of the surrounding atmosphere. This, while reducing congestion, probably tends to expel secretions from the mastoid

cells, with the result that patients not infrequently recover before the final arrangements can be made for mastoidectomy.

As the mastoid begins to reaerify, the X-ray discloses isolated cells in the vicinity of the antrum that are clear as contrasted with the general haziness of the rest of the mastoid. When this appearance is found while the clinical symptoms are at their very height, as is sometimes the case, marked improvement usually follows within the next twenty-four hours. Such cases, however, do not always go on to complete recovery. Those that do not probably contain diploe, either in the cell walls or at the periphery of the cell net, in which an osteomyelitic condition has become so firmly established that reaerification of the pneumatic cells does not suffice to restore the mastoid to normal; while, in the completely pneumatic mastoid, if the infection has not actually destroyed the cell walls, reestablishment of the normal air content is probably all that is required to bring about healing. In addition to purely mechanical factors the beneficial effect of oxygen on the suppurative process is to be considered.

## SUMMARY.

We believe the foregoing demonstrates that variations in anatomy are dependent on the degree of pneumatic development. A study of our complete series of X-ray films is even more impressive in this regard, for they show about every possible gradation, all the way from the completely acellular mastoid to the most widespread pneumatization, while the anatomic variations described and illustrated in this paper are merely the outstanding types of structure. In view of the fact that the structure of one mastoid is likely to vary so widely from that of another, it seems self-evident that in every serious middle ear infection the anatomy of the mastoid in question should be considered along with the clinical evidence in the case. Our textbook descriptions of the pathology and the symptomatology of mastoiditis together with the indications for operation are all based on the typical mastoid. As there is apparently no such thing as a "typical" mastoid, it would seem as though these descriptions should be revised so

as to conform more closely to the main types of mastoid structure as they actually occur.

We believe that Wittmaack's description, both of pneumatization and of types of structure, provides a most satisfactory basis for the interpretation of mastoid anatomy. However, we do not wish to give the impression that we accept his theories and conclusions unreservedly, for one thoroughly familiar with his work will see that this paper is based chiefly on his observations, with but incidental reference to his theories and conclusions. We are fully aware of the objections that can be raised against the latter, but to reject the entire work for this reason alone seems to us most illogical, for in passing judgment on its worth three factors should be considered. First and foremost, do these observations and investigations give us the actual facts as regards complete and incomplete pneumatic development or, as Wittmaack expresses it, regarding normal and pathologic pneumatization? As to this, a review of the literature and consultations with several eminent embryologists have disclosed, so far as we have been able to determine, no investigations whatever in which the histologic processes of pneumatization have been observed and described. We therefore believe that Wittmaack's observations in this regard are not to be rejected unless disproven by subsequent investigations of a similar nature. If we but familiarize ourselves with Wittmaack's work we are then fully able to form our own conclusions as to its practical applications, and whether or not our conclusions agree with those of Wittmaack seems of little consequence. Furthermore, whether we accept his explanation as to the causes of incomplete pneumatization, or whether we can offer a more logical explanation of our own, or whether we hold this question in abeyance seems to us of relatively minor importance.

Without going into purely technical details, it seems well to conclude with a few general remarks as to X-ray examination. If this is to be of any real use to the otologist, it is obvious that it must be adequate as well as technically perfect. We ourselves do not believe that the structure of any mastoid, with the possible exception of infants, can be accurately determined by means of one flat plate, no matter how projected

or how perfect the technic. After a thorough trial of the projections devised by others as well as those devised by ourselves, we believe that an adequate examination requires three plates for each mastoid; a streoscopic set in the usual angle with the two meati superimposed and a flat plate in the occipital projection. The former shows both the structure and general relations of the mastoid in three dimensions, while the latter discloses whatever cell net be present in the mastoid proper, much the same as would a vertical cross section in the sagittal plane. If conditions be such that but one plate can be taken, we have found the occipital projection to be the most useful, especially where repeated examinations are indicated. If double intensifying screens and films be used the radiation otherwise required is so reduced that repeated examinations can be made without danger.

While in nowise minimizing the pathologic conditions that may be disclosed by X-ray examination, we are firmly convinced that focusing the attention solely on this feature has been a potent means of discrediting mastoid radiography in the eyes of many otologists; for, as a matter of fact, the well trained otologist does not need to have the radiologist make his diagnoses or tell him when to operate. We do believe, however, that regardless of age or type of structure, an accurate foreknowledge of mastoid anatomy can be obtained by means of the X-ray and by this means only. Given this information, it is possible to determine the grade of development and to form some conception of the histologic structure of the individual mastoid. These questions then cease to be purely academic, but on the contrary, if brought to bear on the everyday problems of mastoid disease, it would seem as though diagnosis, prognosis and the establishment of indications for operation would be placed on a more scientific, less empiric basis than is otherwise possible.

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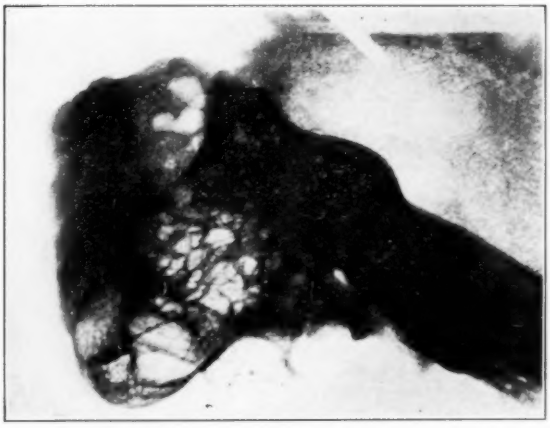

FIGURE 1.
Large cell in tip. Incomplete trabeculation, simulating broken-down and frilled-out walls.

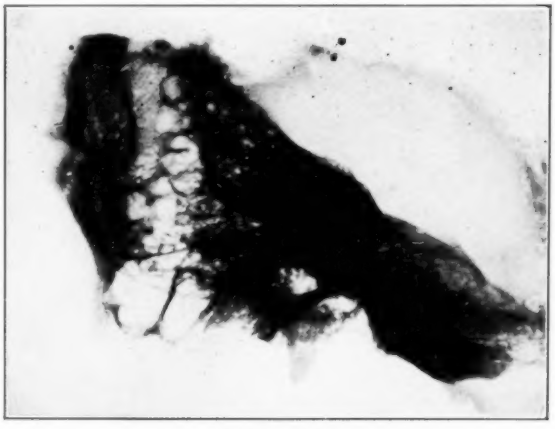

FIGURE 2.
Same condition as Figure 1, somewhat more pronounced.

FIGURE 3.

Mrs. H. Examined in 1917, three and one-half years after an acute mastoid infection. Recovery without operation. Considerable density posteriorly, in midst of which are scattered scraps of pneumatic cells.

FIGURE 4.

Mrs. H. Left normal mastoid, showing completely pneumatic type.

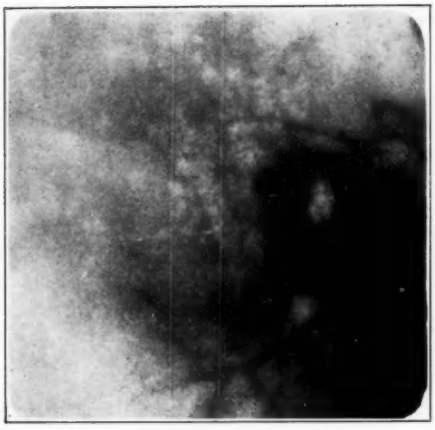

FIGURE 5.

Mrs. H. Right mastoid, examined in 1922, five years after Figure 3. Definite increase in cellular structure.

FIGURE 6-A.

Completely pneumatic mastoid, projected in usual manner with internal and external auditory meati superimposed. Note how petrous pyramid is projected backward, obscuring region of antrum. (Arrow.)

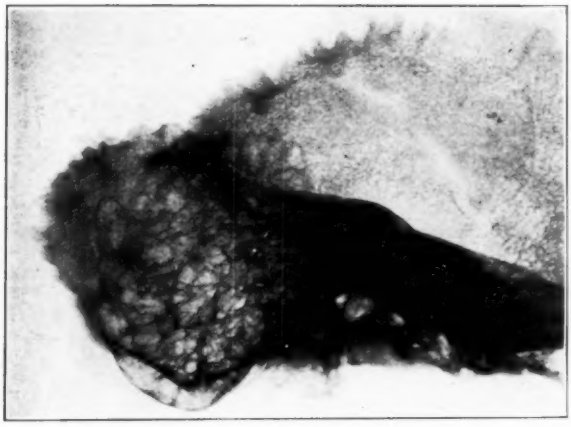

FIGURE 6-B.

Same mastoid, taken with occipital projection. Pyramid is projected forward over tympanic cavity and temporomandibular joint. Antral portion of mastoid unobscured. Note fine meshed cell net in this region.

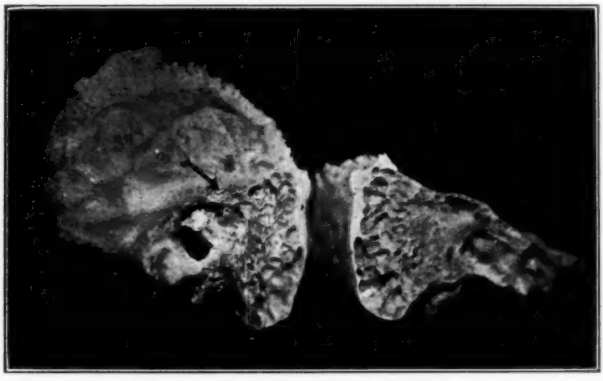

FIGURE 6-C.

Same bone in vertical cross section, sagittal plane. Note that occipital projection (Fig. 6-B) shows approximately same cell distribution as this sectioned bone. Arrow points to fine meshed cell net in outer antral wall, resembling diploe.

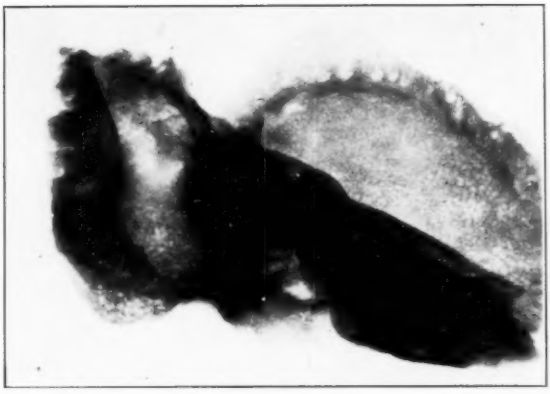

FIGURE 7-A.

Occipital projection showing complete absence of pneumatic cells. Lateral sinus very prominent, due to superficial and forward position.

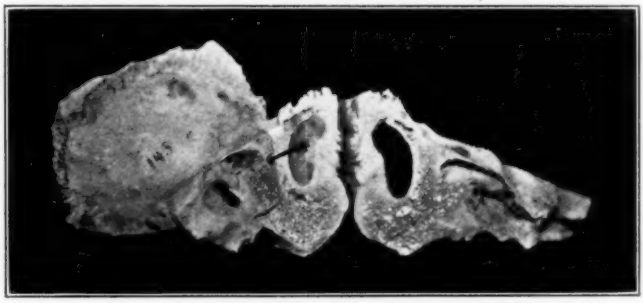

FIGURE 7-B.

Same bone sectioned. Arrow points to compact bone separating antrum from diploe. Several tiny depressions are seen in outer antral wall.

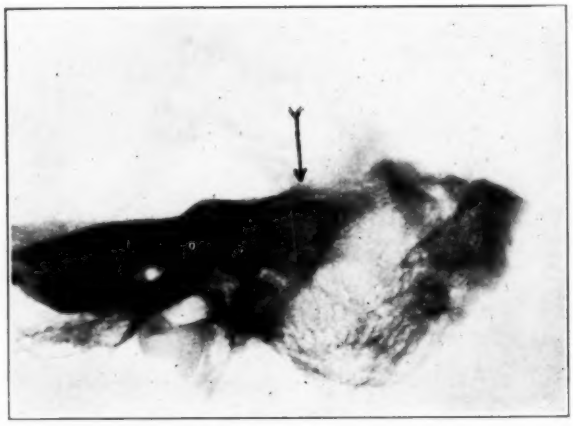

FIGURE 8-A.
Occipital projection, showing a fine meshed cell net (arrow) in immediate vicinity of antrum.

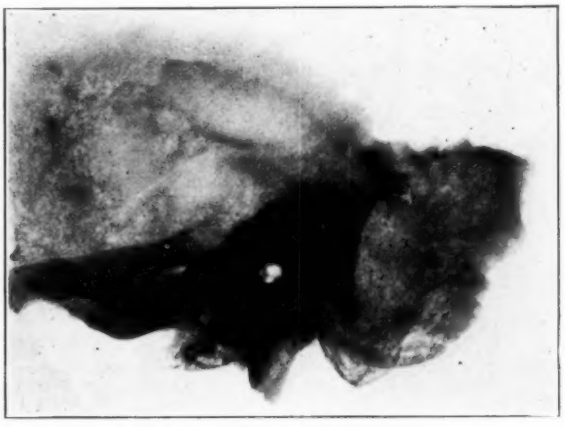

FIGURE 8-B.
Regular projection. Cell net almost entirely obscured by backward projection of petrous pyramid.

FIGURE 8-C.
Sectioned bone. A—Fine meshed cell net about antrum. B—Compact bone separating antrum from diploe.

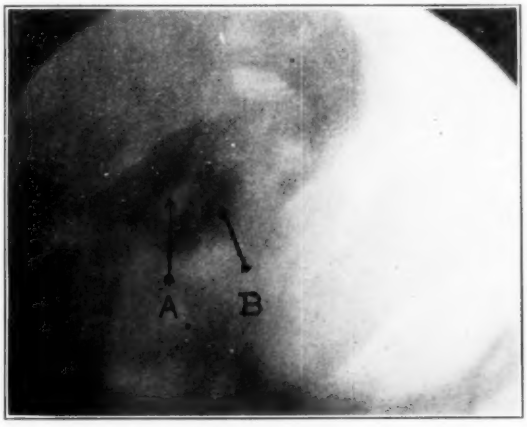


FIGURE 9-A.
Child 22 months old. Regular projection. A—Tympanic cavity. B—Pyramid obscuring antral region.

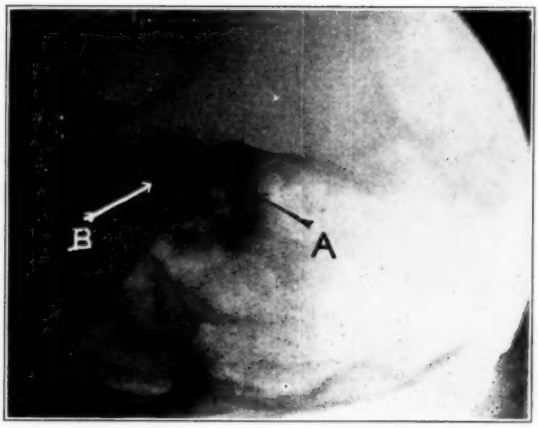


FIGURE 9-B.
Same child, occipital projection. A—Small but definite cell net seen about antrum (arrow). B—Pyramid projected forward.

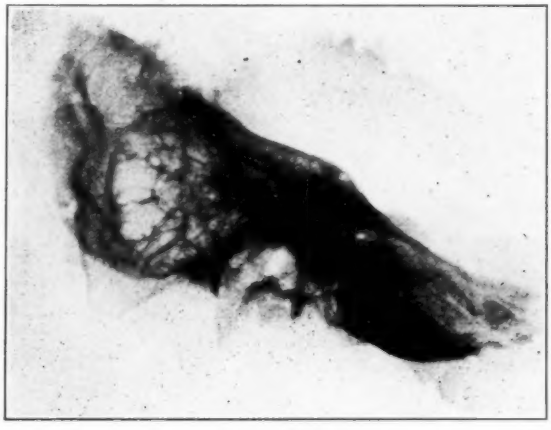

FIGURE 10.
Occipital projection. Upper two-thirds shows typical pneumatic cell net, with fine meshes in region of antrum widening out toward periphery. There is some limitation of cell net posteriorily, while tip is entirely diploetic. Note thickening of peripheral cell walls.

FIGURE 11-A.

Usual projection. Tip completely pneumatic. Considerable limitation of cell net in upper half.

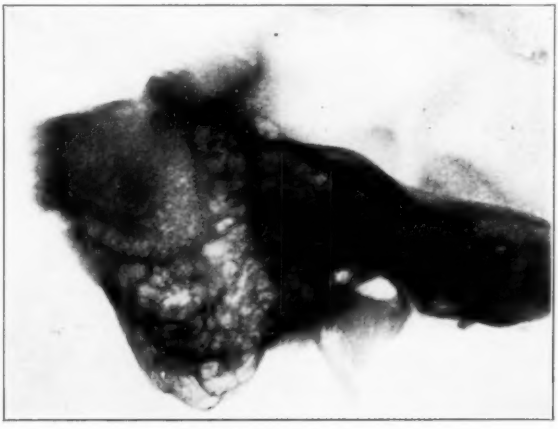

FIGURE 11-B.

Occipital projection. Partial development of upper half seen more clearly. This demonstrates superiority of occipital projection in depicting region of antrum.

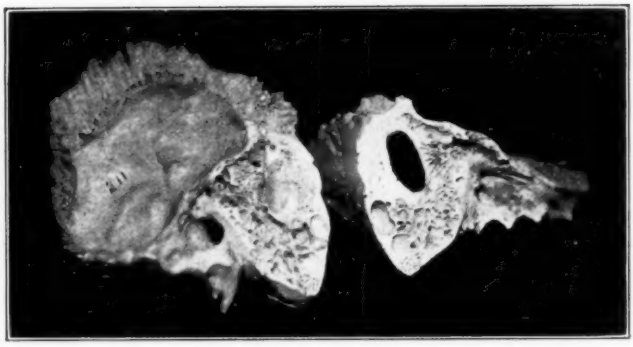

FIGURE 11-C.

Vertical cross section, sagittal plane. Again demonstrates that occipital projection brings out cell net practically as well as section of bone. (This bone was sectioned further, but no additional pneumatic cells were found in upper half.)

FIGURE 12.

Cell net very much limited in extent. All cells are small, without thickening of peripheral walls. Lateral sinus superficial and forward.

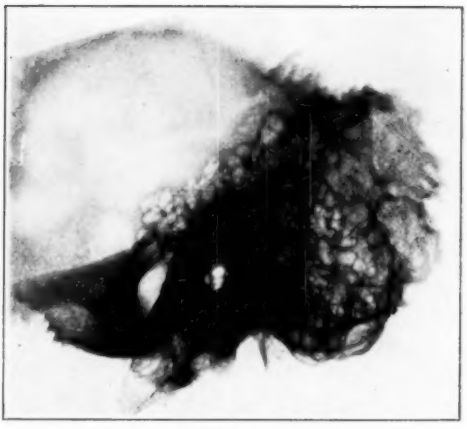

FIGURE 13-A.
Usual projection. Extensive pneumatization, cells reaching through zygoma to temporomandibular joint.

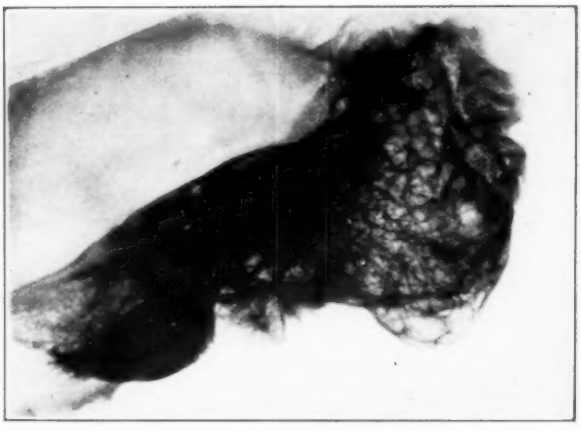

FIGURE 13-B.
Occipital projection. This view does not reveal cells in region of zygoma as well as usual projection.

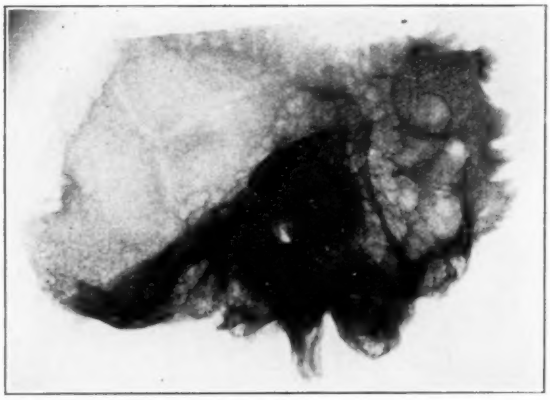

FIGURE 14-A.
Usual projection. Extensive pneumatization of upper portion, while tip is completely diploetic.

FIGURE 14-B.
Occipital projection. Shows sharp limitation of penumatic structure and thickened peripheral walls near tip.

FIGURE 15-A.
Occipital projection. Cell net shows limitation posteriorly. All cells are thickened.

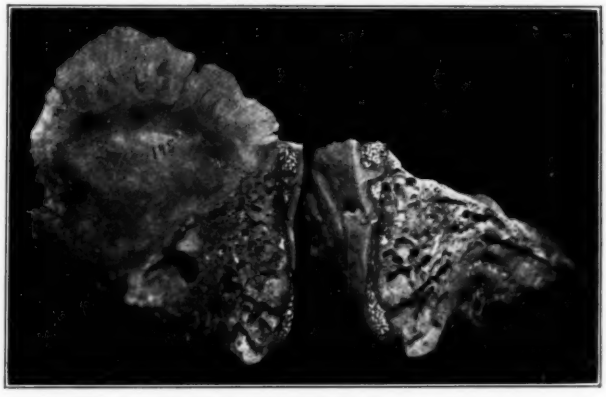

FIGURE 15-B.
Sectioned bone, showing same condition.

L.

# THE INCIDENCE AND HISTOPATHOLOGY OF BONE AND CARTILAGE IN THE TONSIL.*

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Ann Arbor.

To the clinician, the occurrence of larger or smaller nodules of bone and cartilage in the tonsillar capsule and stroma, palpable to the examining finger or mechanically interfering with operative procedures, has always been a matter of great interest. Similarly, the histopathologist, whose microscopic sections reveal an altogether unexpected incidence of such nodules, many of them so small that they could not be detected otherwise, finds that their occurrence and histogenesis throw light upon an important part of the pathology of the tonsillar region. Although Barnes[2] dismisses the finding of bone and cartilage in the tonsil with a word, the increasing bibliography upon this subject, and especially the persistent appearance in the same journals of successive articles dealing with it, bear witness to the interest which it arouses. The latter point may well be illustrated from the Annals, in which there have appeared the papers of Grosvenor[9] (1908), Kelsey[14] (1911), Grove[10] (1916), Albert and Douglis[1] (1917) and Forman[7] (1918). That such a sequence of reports seemed desirable and necessary has been due to the fact that no one of them has decisively established either the frequency of occurrence of such nodules of cartilage and bone or the relative parts played by chronic inflammation and inherent tissue potentiality in their genesis.

It seems unnecessary to give an extended review of the literature of bone and cartilage in and about the tonsil since the first reported cases of Orth[20] in 1893. A bibliography is appended and to its more important items reference will be made at appropriate points. However, there are three important aspects of this literature to which general reference must

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here be made, since they mark stages of progress in the growth of this subject. Many of the earlier papers report the occurrence of gross islands of bone and cartilage, such as interfered with the removal of the tonsil, and attention was paid to the continuous or discontinuous relationship of these to the styloid process. In another, and in part simultaneously appearing group of papers, we find case reports of a few instances, perhaps only one or two, in which bone or cartilage was discovered in the course of microscopic examination of the tonsil. In the erroneous belief, based upon a limited experience, that such a finding was highly unusual, a single observation was considered worth reporting. In the third place, from the standpoint of etiology, the earlier literature was highly controversial, and arguments for and against metaplasia associated with chronic inflammation as contrasted with developmental disturbances and growth of "embryonal rests" play an important part; while in more recent papers increasing attention is given to the embryologic history of the parent tissues and their inherent powers of proliferation and metaplasia.

Material and Method.—About 15,000 pairs of faucial tonsils have now been examined microscopically in the pathologic laboratory of the University of Michigan. These have been fixed routinely in formol, into which they are placed in the operative clinics. Upon receipt in the laboratory they have been bisected longitudinally in a plane at right angles to the oropharyngeal surface, and one-half of the tonsil has been embedded in paraffin for sectioning, regardless of its size. The sections have been cut from the surface produced by the bisection, thus giving the largest possible area of tonsil tissue for examination and one which presents at the same time surface mucosa, longitudinal and cross sections of the ramifications of many of the crypts, the septa between crypt systems and the capsular structures. Such an enormous material can not be examined in serial sections, but experience has shown that pathologic changes of importance will rarely be missed in a systematic field-by-field examination of a few sections oriented according to the method developed in this laboratory. Hemalum and eosin staining has been employed routinely, with special stains as indicated.

In a majority of the cases the original pathologic diagnoses

were made by Dr. A. S. Warthin, and upon this accumulated data I have been permitted to draw freely. For the statistical purposes of this article, however, the writer has made a systematic field-by-field reexamination of the sections from 1,000 consecutive pairs of tonsils with special attention to the points here considered. It so happened that in these 1,000 consecutive cases there were 76 in which the age was not known. In order to have age incidence data for 1,000 cases available, 76 additional consecutive cases of known age were included. Two series of cases are therefore considered in appropriate sections of this report: 1,000 consecutive cases and 1,000 consecutive cases of known age. As will be pointed out, there were slight differences due to chance distribution in the bone and cartilage incidence in the two series. In a very few cases one tonsil only was received, but this occurred so infrequently that it need not be considered in a mathematical analysis of the material.

The large number of cases here considered, far in excess of any other report upon this subject, renders the factor of chance distribution negligible; and the consecutive nature of the series chosen for statistical study nullifies any criticism based upon the fallacy of random sampling. Thus the two forms of error, most often met in medical statistics, are avoided. There are, however, serious sources of error, inherent in the method itself and of chief importance in their effect upon the percentage of incidence of bone and cartilage. These all operate in the same direction, depressing the ascertained incidence below the actual fact, so that all figures here presented represent a minimum value demonstrably lower to an unknown degree than the true incidence. For example, bone and cartilage may be left behind in the course of removal of the tonsil, or may tear out in the cutting of the section through their greater resistance to the microtome knife, the resulting ragged artefact revealing nothing to show what may have been present. Moreover, in the examination of a relatively small number of sections, one or two, from each tonsil, those islands of cartilage and bone which are located in the more anterior and posterior portions of the capsule are necessarily missed. This is not as effective a source of error as might be imagined, for the tonsils having bone or cartilage in such situations usually

have multiple nodules elsewhere as well. Nevertheless, the inability to study serial sections in dealing with so extensive a material cannot do otherwise than produce subminimal values for incidence. The writer is fully aware of certain other sources of error inherent in dealing with material in which a large number of variable factors are simultaneously operative. It may well be that the presence of bone and cartilage in the tonsil favors those conditions which bring the patient to the otolaryngologist, or that, on the other hand, such conditions may have had some causal relationship to the production of the bone and cartilage. In that connection it must be remembered that the only claim made for the figures here set forth is that they represent a minimum incidence value for bone and cartilage in tonsils removed at operation. It is the belief of the writer that the incidence in the general population, if obtainable by the same method, would not be far different from the value here ascertained, a belief based upon the relatively slight manifestations of disease which have been deemed sufficient to warrant tonsillectomy.

INCIDENCE.

General Incidence.—The tonsils from 1,000 consecutive tonsillectomies examined histologically show cartilage or bone, or both, in 209 cases, an incidence of 20.9 per cent. Few similar series are offered for comparison in the literature. In 50 tonsils from 37 different individuals Reitmann[22] found bone or cartilage in 17, or 34 per cent. Apparently, these were from 14 individuals, an incidence of 38 per cent on a basis similar to ours. In the material from 412 autopsies Lubarsch[16] found bone and cartilage 65 times, or 15.77 per cent. Maclachlan[17] collected eleven cases among 350 pairs of tonsils, an incidence of a little over 3 per cent. Forman[7] found only one specimen containing cartilage in the examination of 290 consecutive pairs of tonsils. This remarkably small incidence may have been associated, he suggests, with an almost equally low percentage of tuberculosis. From our experience, we know that some other factor must have been operative, for the coincidence of those two conditions is no more frequent than chance association demands. It must be understood that for reasons which have already been given the incidence of 20.9 per cent determined by us is a minimum figure. Also, in any series,

the age distribution in the entire group will be found to greatly influence the incidence. Comparisons can be made profitably only when such factors are considered.

In the 209 cases positive for bone and cartilage, in the series here reported, 53 showed cartilage in both tonsils, while in 156 it was found in but one. Similarly of the 59 with bone, 19 showed bone in both tonsils and 40 in only one. The 59 cases in which bone was found are all included in the 209 showing cartilage. In other words, while bone was never found without some cartilage occurring also, it was found in but 28.3 per cent of the cases showing cartilage.

Incidence as Influenced by Sex.—In the 1,000 consecutive pairs of tonsils there were 579 from male patients and 421 from female patients. The former yielded 126 cases showing bone or cartilage, an incidence of 21.76 per cent. The 421 cases in females yielded 83 positive cases, an incidence of 19.71 per cent. The difference between the percentages of incidence in the two sexes is without significance, falling well within the range of error inherent in the method of selection and analysis of this material.

Incidence as Influenced by Age.—In the 1,000 consecutive tonsil cases of known age (a slightly different series than the one considered for general and sex incidence) 205 cases showed the presence of bone or cartilage or both. The youngest positive case was at age three, with three positive cases at age four and five positive cases at age five. The oldest positive case was found at age 59, with two at 56 and two at 55. Since this material was surgical in origin and not derived from the autopsy table, the extremes of life have but a meager representation. The incidence just given is therefore illustrative only of the fact that bone and cartilage may be met with at any age.

The average age of the 1,000 cases was 18.722 years, while the average age of the cases showing cartilage and bone was 24.263. That advancing years increase the incidence is still more evident when it is noted that the average age of the 795 negative cases was 17.295 years, making a difference of almost exactly seven years in the average ages of the two groups. Moreover, it must be borne in mind that those in the second, third and fourth quinary periods of life constitute the group

from which the larger proportion of the tonsillectomies is done. If consecutive cases are considered, this period, therefore, will have undue weight in determining the average age, provided the incidence of bone and cartilage is not fairly uniformly distributed throughout the years of life, and it is evident from the average ages of the positive and negative cases in this series that further analysis as to age distribution is indicated for the same reason.

If, then, the cases be segregated according to age periods and the percentage of bone and cartilage incidence be determined separately for each period, the following table can be constructed:

TABLE I.

| Age Period | Total Cases | Positive | Negative | Incidence |
|---|---|---|---|---|
| 1— 5 | 75 | 9 | 66 | 12% |
| 6—10 | 236 | 23 | 213 | 10 |
| 11 -15 | 187 | 20 | 167 | 11 |
| 16—20 | 133 | 32 | 101 | 32 |
| 21—25 | 120 | 30 | 90 | 33 |
| 26—30 | 90 | 34 | 56 | 61 |
| 31—40 | 96 | 35 | 61 | 57 |
| 41—73 | 63 | 22 | 41 | 54 |
| | 1,000 | 205 | 795 | |

The change of incidence is shown graphically in Figure 1, in which the values of the final column of the table are plotted as if the cases were concentrated in the terminal year of each age period. It will be noted that in spite of the large number of cases considered and the consequent reduction in error, the incidence of bone and cartilage remains practically constant for the first 15 years of life. It then rises rapidly in the next fifteen years to a level five times as high, where it remains without further increment, the slight downward tendency in the later years probably being an inaccuracy introduced by the greatly lessened number of cases.

HISTOPATHOLOGY.

Cartilage and bone are found only in the connective tissue structures of the tonsil, the capsule and the trabeculæ. Apparent exceptions to this rule occur, in which islands of cartilage

are found close to the pharyngeal mucosa or partially included within lymphoid tissue. Careful examination will always show, however, that such islands of bone or cartilage are entirely included within extensions from the capsule and never within the actual substance of the lymphadenoid tissue. (See Figure 8.)

In searching for the occurrence of these substances it is noted that cartilage is more frequently found in association with the small mucous glands of the tonsillar capsule (Figures 2, 4 and 5), particularly the group occurring near the lower pole, than in any other situation. This finding is met with so frequently that the tonsils showing it constitute a special group of cases. In this location it occurs in even very young children, in whom it is by far the most frequent type and in whom it is oftentimes found unassociated with any increase in the general stroma of the tonsil. When thus found, it may be the only nodule of cartilage present. Groups of voluntary muscle fibers are frequently associated with the cartilage and mucous gland alveoli in this type, but it is very rare to find ossification in the islands of cartilage occurring in this situation.

With this group of cases can be contrasted another, occurring more frequently in older patients, although we have seen it a number of times in children under six years. In these there is no special association of the bone and cartilage with the mucous glands, but the association is with an increase of stroma and fibrosis of the supporting tissue. The islands of bone and cartilage are usually multiple (Figure 3), sometimes very numerous and tend gradually to coalesce, forming in extreme cases a ramifying skeletal mass occupying the thickened capsule and trabeculæ. In this type ossification is very apt to occur.

Membranous osteogenesis has not been found in any instance and probably does not occur in the tonsil. Therefore bone is never encountered without cartilage being present also. However, there are several instances in our total series in which the rim of cartilage surrounding an island of bone is so narrow that it might be very easily overlooked if not closely examined. In such cases, the usual progression of connective tissue to cartilage and of cartilage to bone is apparently inter-

rupted, in that the new formation of cartilage practically ceases while bone formation continues until the cartilage is almost, if not quite, exhausted.

In those tonsils in which the supporting connective tissue contains numerous islands of cartilage and bone, all stages in the histogenesis of those tissues may be found at the same time. The process is progressive and continuous, leading from dense connective tissue, microscopically showing little to differentiate it from any other connective tissue, to bone with well developed Haversian canals and a highly vascularized bone marrow. The process may, however, be retarded or checked at any stage.

The connective tissue of the areas in which cartilage is to develop (except those islands of cartilage developing in association with mucous glands) would in most cases be considered to be increased in amount, so that in a descriptive diagnosis such expressions as "increased stroma" or "fibrosis" would be applied to such a tonsil. This connective tissue is, perhaps, somewhat more coarsely fibrillar and shows a slightly greater tendency to form whorls than that ordinarily found. In such an area a very small, rounded localized center appears in which the fibrillar structure is less evident and the eosin staining diminished in intensity. As these changes become more marked, the cells become more rounded, their cytoplasm clearer and the nuclei oval or spherical (Figures 6 and 7). Such cells come to lie in a matrix which is more clear and which, with nearly complete loss of evidence of fibrillæ, becomes somewhat mucinoid, staining bluish with hemalum. In such areas, now closely resembling cartilage, the cells come to have characteristic clear capsular spaces, but show a more uniform distribution and less grouping than is usually seen in cartilage in other situations. This indicates relatively little growth of the islands of cartilage through proliferation of their fully differentiated cells. In full accord with this is the evidence of peripheral extension, for the process of transformation of connective tissue into cartilage may at times be found in all stages in the surrounding zone. In other instances the surrounding connective tissue becomes somewhat compressed and laminated and forms a definite capsule or perichondrium for the cartilage nodule. While no vessels are found in the substance of the

cartilage, the connective tissue capsule may be well supplied. The formation of the cartilage is therefore a process of direct metaplasia.

Calcification of the cartilage is the first step toward bone formation. The cartilage cells usually become larger and more globose while lime salts are deposited in a network of delicate trabeculæ around and between them. This deposit of lime salts in the matrix continues until a heavy and sometimes coarsely granular mass is formed, staining a deep purplish blue with hemalum and eosin (Figures 9—12). True bone is formed only with the destruction of this primary calcification by a vascular osteoclastic and osteoblastic tissue, which is derived in part, and perhaps entirely, from the surrounding capsular structures. The source and nature of the cells concerned in this process present the same difficulties of interpretation as in endochondral ossification elsewhere, a process which the further elaboration of true bone in the tonsil resembles in every particular. In a number of instances we have seen the usual convex surface of the nodule of partially calcified cartilage indented by a fibroblastic and angioblastic bud apparently invading it from the vascular capsule. This is apparently the method by which the osteoblastic tissue reaches the interior of the nodule. Then by repeated phases of formation and destruction the bony area enlarges peripherally, but soon comes to have a fully developed marrow space in the interior, which may contain a fatty, highly vascular marrow (Figures 13 and 14). The surrounding bone shows typical lacunæ, leaving no room for the doubt expressed by Grove[10] as to the occurrence of true bone in the tonsil.

Although we have seen many instances of the type of cartilage occurrence in which there is a solitary nodule or barlike mass in association with the mucous glands and without noteworthy increase in stroma, we have never observed the stages of bone formation in these cases. We must conclude that this cartilage differs in certain respects from that found in the other type and that one of these differences is its tendency to remain unaltered for many years, failing to show the primary calcification which in other situations is the forerunner of osteogenesis.

There is no fixed association of cartilage and bone in the

tonsil with tuberculosis or any other specific infection. We have now seen several hundred examples of tonsillar tuberculosis, and we find that such tonsils show cartilage and bone in approximately the same proportion as the nontuberculous tonsils of patients of the same age groups. There is a definite association of the type in which multiple nodules of cartilage and bone are found, with a well marked increase in the connective tissue of the capsule and trabeculæ, but it is noteworthy that many cases with equally marked fibrosis show no bone or cartilage. Kellert[13] concluded that there is no certain relation between cartilage in the tonsillar capsule and inflammation.

## DISCUSSION.

In discussing the results of this analysis it will appear that they support the belief that the occurrence of bone and cartilage in the tonsil is not due to any misplacement of cartilage or bone forming cells or to any other disturbance of development in the sense in which that expression was used by the earlier writers upon this subject. The process is essentially one of metaplasia, but is made possible by the inherent nature of the tissue concerned and is furthered by the occurrence of a chronic fibrosing inflammatory reaction.

For the earlier arguments between those believing the process one of metaplasia and those insisting that such formations could come only from "rests," the papers of Rückert,[24] Carter,[3] Theodore,[27] Cary,[4] Albert and Douglis[1] and others may be consulted. Nösske,[19] in 1903, had come the nearest to an accurate statement of the genesis of tonsillar bone and cartilage when he wrote that in addition to inflammatory and retrogressive processes a sort of local predisposition plays an etiologic rôle; that the connective tissue of the tonsil is endowed with a special cartilage and bone forming capacity which can depend upon a developmental "moment" without the necessity of assuming any embryologic disturbance.

In a recent paper Lipshutz[15] emphasizes the importance of a thorough appreciation of the part played by the structures derived from the second branchial bar in connection with the occurrence of either continuous or discontinuous bony or cartilaginous masses along the course of the stylohyoid ligament. In its origin from the second branchial pouch the tonsil de-

rives its supporting tissue from the second and third branchial arches. Themselves cartilaginous, the branchial arches contribute a tissue which possesses to an unusual degree the power of cartilage and bone development. We find that the observed facts are in complete accord with this explanation. The tonsils of young children which show an incidence of but 10 per cent or 12 per cent of bone and cartilage, if allowed to remain in position twenty years or so longer would show an incidence of bone and cartilage of 50 to 60 per cent. Yet one cannot tell by microscopic examination which of the tonsils of very young children would, and which would not, produce cartilage and bone if left in. There are no "rests" of cartilage or bone cells. Moreover, the inherent power to form cartilage and bone is not possessed by the tonsillar supporting tissue to the same degree in all cases. Some tonsils show marked fibrosis without any cartilage.

The method of osteogenesis in the tonsil is that which would be expected in derivatives from the branchial bars. There is no authentic evidence of intramembranous ossification in the tonsil, and we believe that the instances in which bone seemed to be present without cartilage are to be explained by a complete or nearly complete replacement of the cartilage by bone without the continuous production of new cartilage at the periphery of the nodule, as is usually the case.

The occurrence of nodules of cartilage at the site of the developing tonsil in fetuses, as described by Lubarsch[16] and others, by no means militates against the view here presented. In the first place, there is a legitimate doubt as to the exact location and relationship of the nodules found in such fetuses, due to the relatively late development of the tonsil itself. Even if found at an early age, such nodules are still subject to the same explanations as in later life, for the cartilage forming potentiality may become effective at any age.

Is the difference between an incidence of 10 per cent at 10 years of age and an incidence of 60 per cent two decades later to be explained by the one fact alone that time has elapsed for the development of cartilage and bone nodules in a tissue having that potentiality? It scarcely seems possible that so abrupt a change could result if this were the sole factor. Rather it would seem that the proliferation of new connective tissue

associated with the healing of repeated inflammatory processes itself awakens and stimulates the latent power. One is forced to this conclusion by the frequent association of much scar tissue and a general increase of connective tissue with multiple nodules of bone and cartilage in the very young as well as in the old. That cases occur without much or even any diffuse fibrosis serves but to demonstrate more completely the importance of the histogenetic inheritance of this tissue. Particularly is it true in the type of case in which a single bar of cartilage is found in association with the mucous gland and often also with a few voluntary muscle fibers that chronic inflammation plays an insignificant part. This fact is in accord with the observation that this is the more common type in very young children. It must be true that the supporting tissue of this area is prone to produce cartilage without the aid of stimuli dependent upon or associated with any known pathologic condition.

## CONCLUSIONS.

1. In 1,000 consecutive pairs of tonsils, out of 15,000 examined, the minimum incidence of cartilage and bone, as determined by the systematic microscopic search of a limited number of sections from each tonsil, was found to be 20.9 per cent.

2. Bone was never found without at least some cartilage and occurred in 28.3 per cent of the 209 cases in which cartilage was found.

3. The incidence was not significantly affected by a segregation of the cases as to sex.

4. The average age of cases showing cartilage or bone was 24.263 years; of those which were negative, 17.295 years.

5. The incidence of cartilage and bone remains at a nearly constant level up to puberty. It then rises rapidly to a high level at age period 26 to 30, after which it appears to remain practically constant. The period of most rapid increase in incidence is thus coincident with the period of most active osteogenesis in the skeletal development of the individual and is therefore apparently subject to the same laws as those governing general bone development.

6. Cartilage and bone are found only in the connective tissue structures of the tonsil, where they occur in two somewhat distinct types.

7. In one type the microscopic sections show a single mass of cartilage in close association with a mucous gland. We have not observed bone formation in this type. It is possible that the occurrence of cartilage in this situation is within the range of anatomic variation and has no pathologic significance.

8. In another type multiple foci of cartilage and bone occur throughout the capsule and trabeculæ, usually associated with considerable increase in connective tissue.

9. Cartilage is formed by a process of progressive transformation (direct metaplasia) of the connective tissue, while bone in the tonsil always arises in such newly formed cartilage by processes wholly comparable to endochondral ossification elsewhere.

10. Disturbance of development in the sense of "rests" of cartilage and bone cells cannot explain the occurrence of these tissues in the tonsil.

11. The inherent predisposition of the connective tissue of the tonsil to form cartilage depends upon its origin from branchial arch structures. This latent tendency becomes especially effective under the influence of chronic inflammation.

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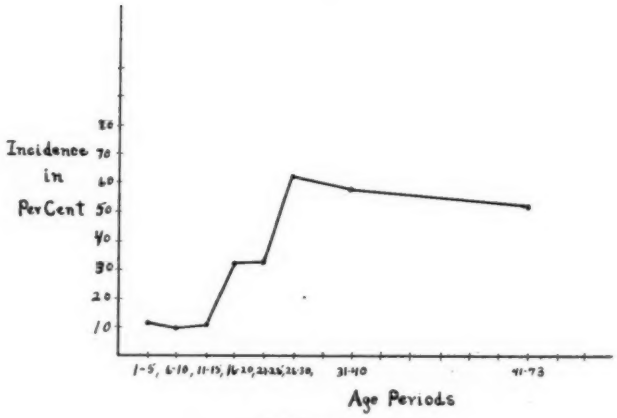


FIGURE 1.

Curve of age-incidence of bone and cartilage in the tonsil, based upon 205 consecutive positive cases of known age. The percentage of incidence is plotted against five year age periods. The maximum incidence is reached in the age of 26 to 30, after which there is practically no change.

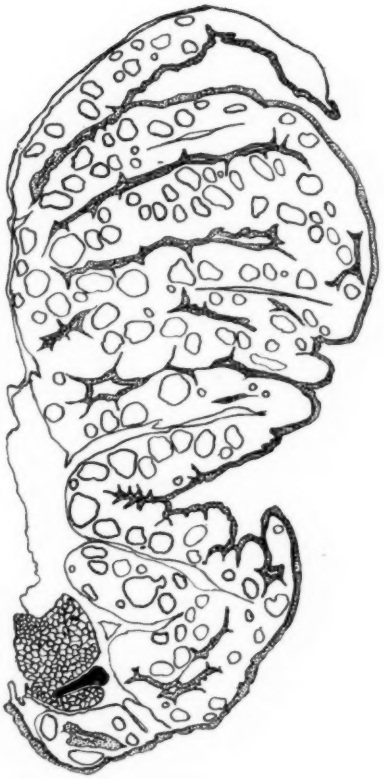

FIGURE 2.

Outline tracing of actual section of tonsil cut through the greatest vertical plane, to show the location of the cartilage (in solid black) as very frequently found near the mucous glands. The more frequent type in young children and unassociated with increase in stroma. This is probably a normally occurring structure in many individuals.

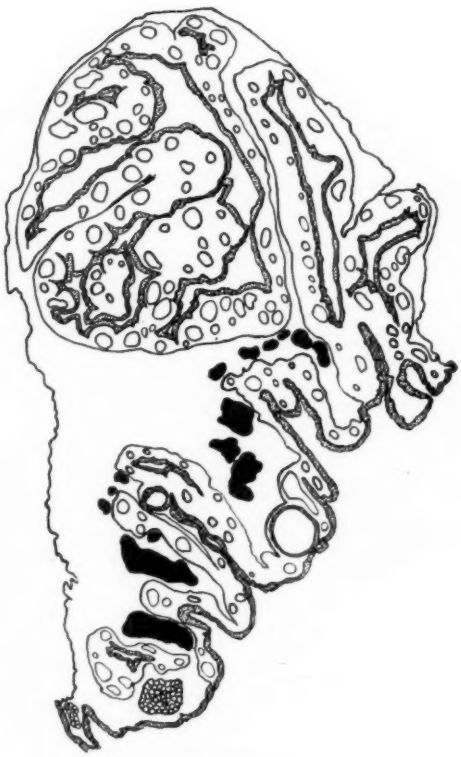

FIGURE 3.

Outline tracing of actual section of tonsil to show the distribution of bone and cartilage (solid black) in the group of cases in which the association is not with the mucous glands but with increase of stroma and fibrosis of the supporting tissue.

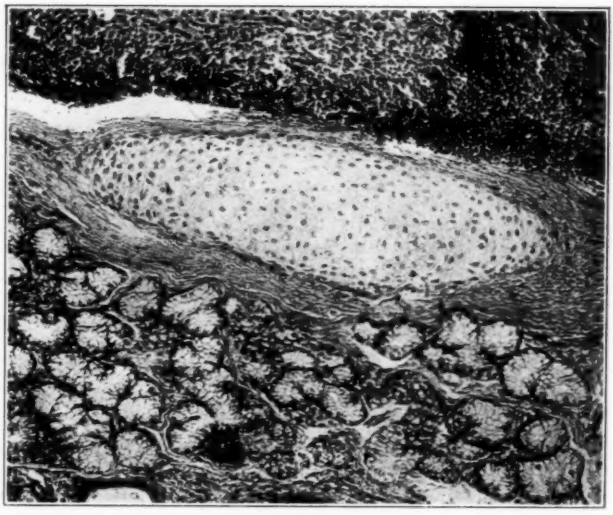

FIGURE 4.

Photomicrograph (hemalum and eosin stain, Zeiss obj. "B," no ocular) showing nodule of cartilage occurring in association with mucous gland alveoli. The more frequent type in young children.

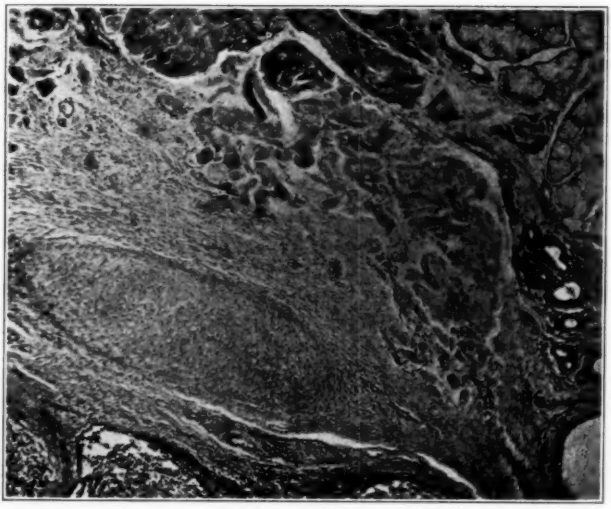

FIGURE 5.

Photomicrograph showing nodule of cartilage occurring in association with mucous glands and a group of voluntary muscle fibers. Another example of the type first described in the text.

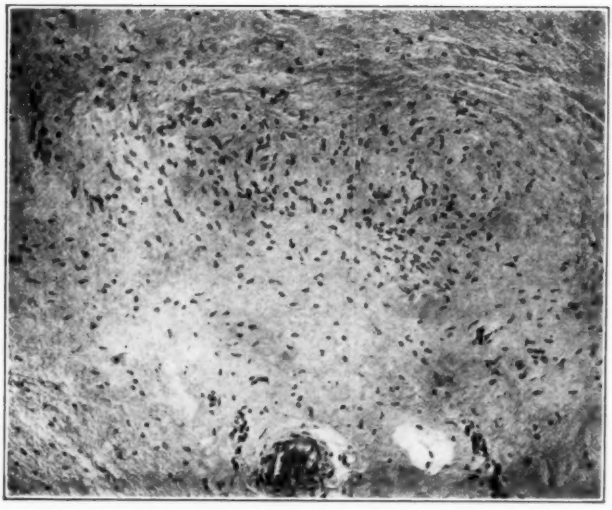

FIGURE 6.

Photomicrograph showing an early stage in the direct metaplasia of fibrous connective tissue to cartilage in the stroma of the tonsil. This tonsil showed the simultaneous development of multiple cartilage nodules. These nodules show no relationship to the normally occurring mucous glands. The type more commonly found in adult patients.

FIGURE 7.

Photomicrograph of somewhat more advanced cartilage formation than that shown in Figure 6. Many of the cells have now become globular and the cartilage character of the nodule is well established.

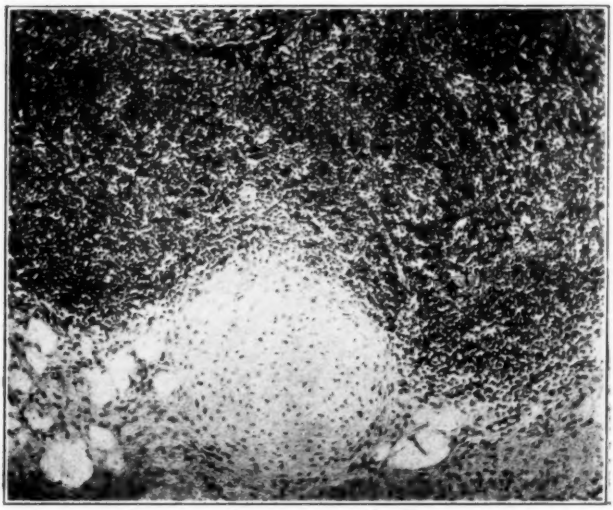

FIGURE 8.

Photomicrograph showing a well developed nodule of cartilage in the stroma of the tonsil. It is so close to the lymphoid tissue that it intrudes upon it, although actually situated entirely in the stroma. This appearance has at times been incorrectly interpreted as cartilage developing in the lymphoid tissue.

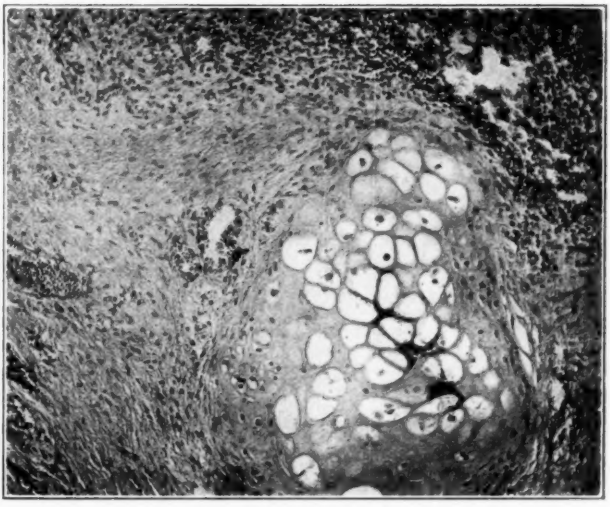

FIGURE 9.

**Photomicrograph showing early stage in the calcification of cartilage in the tonsil. The first step toward bone formation.**

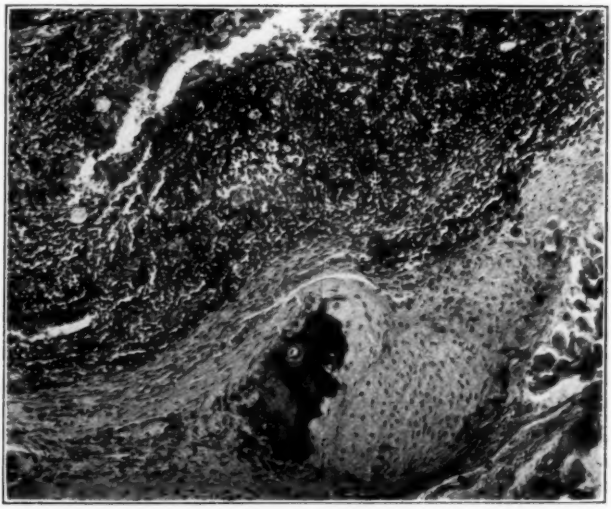

FIGURE 10.

Photomicrograph of more advanced calcification of the cartilage in the tonsil.

FIGURE 11.

Photomicrograph showing in a single field several stages of transformation of cartilage into bone. Early and later stages of calcification of cartilage, true bone formation, ingrowth of vascular tissue to form the marrow.

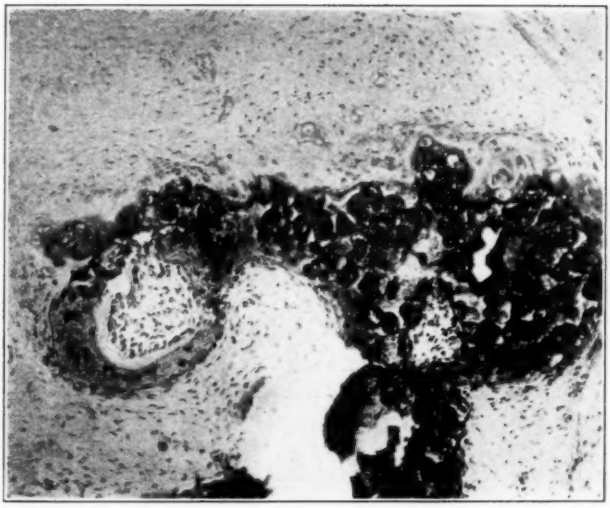

FIGURE 12.

Irregular calcification of cartilage and replacement of the calcified cartilage by true bone with included bone cells and a vascular marrow.

FIGURE 13.

**Photomicrograph of advanced bone formation in the tonsil. Bone cells and a very abundant marrow are present. Some of the calcified cartilage still persists.**

FIGURE 14.

Photomicrograph of advanced bone formation. Process now practically complete. Typical bone cells and abundant vascular and fatty marrow. Canal through bony wall for entrance of vessels to the marrow and endosteum.

LI.

# NOTE ON THE RELATION OF THE OPTIC NERVE TO THE LAST POSTERIOR ETHMOID CELL.*

By S. B. Westlake, M. D.,

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The clinical relations of the eye to the nose have been made the subject of much study in the last few years. The variety of these clinical manifesations have been almost endless; in fact, it would appear that almost every eye affection has been attributed to a nasal condition. We are, however, wandering away from reflex action as responsible for many of these conditions and are searching for definite pathologic etiology. The sinus relations have been the basis of most of the investigation of this subject, whether it be anatomic, physiologic or bacteriologic. When these have been considered inadequate by way of explanation, focal infection has been held accountable.

But it is being recgonized that if we are to achieve any great success in this study, and if we are to have a definite pathologic basis, we must have more essential anatomic data.

The value of definiteness in this respect was beautifully shown by Van der Hoeve at the meeting of the American Academy of Ophthalmology and Otolaryngology in September, 1921, in the report of a case. The symptoms in this patient had persisted in spite of extensive operation. It was only after an ethmoid cell had been located by a careful X-ray examination and exenterated, that the eye symptoms entirely disappeared.

Further studies in the anatomy of the paranasal sinuses are therefore necessary.

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H. W. Loeb ("A Study of the Anatomic Relations of the Optic Nerve to the Accessory Cavities of the Nose," ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, St. Louis, 1909) in his investigation on the relation of the optic nerve to the paranasal sinuses, made some dissections which showed that the optic nerve usually has a minimal relation to the ethmoid labyrinth.

His series of fifteen heads sectioned horizontally showed that in 28 of the 30 instances the optic nerve came into close relation with the last posterior ethmoid cell (occasionally there were two) only at the posterior external superior angle of the cell, from which point it left the labyrinth region to attach itself to the globe. In two instances, however, where the sphenoid was replaced by an ethmoid cell, the nerve's course was along the external wall of the cell for a considerable distance.

To continue these studies with a view of confirming or disproving his observations, he suggested that I should prepare new dissections which would make it possible to study the external cranial wall of the ethmoid cells and sphenoid sinuses. Accordingly seven heads were decalcified and treated according to the technic described by him and, after the cranial vault above the level of the sella had been cut away and the brain removed, the superior and external bony walls of the sinuses were cut away, as far as possible maintaining the mucosa intact and the optic nerve prepared from the periorbita. The results are as follows:

Head I (Fig. 1).—Both sphenoids, fairly large and uniform in size, shown cut open, with mucosa lost. Ethmoid cells on the left side shown with mucosa of external wall intact, that of the roof being cut away. Optic nerve courses along the external wall of the sphenoid, just below its roof, and the superior posterior external angle of the last posterior ethmoid cell from which it proceeds externally to the globe.

Head II (Fig. 2).—The optic nerve has been cut away, exposing the mucosa of the maxillary roof and the external walls of the ethmoid cells and sphenoid sinus.

Head III (Fig. 3) (Right).—This most successful preparation shows the complete mucosa of the superior and external wall of the maxillary sinus and of the external walls of the ethmoid cells. The mucosa of the sphenoid was lost in the preparation, but the bone cavity itself is shown in relation with the optic nerve and adjacent ethmoid cells. The optic nerve presents the usual relation with the last posterior ethmoid at its posterior, superior and external angle.

Head III (Fig. 4) (Left).—In this preparation the dissection was not so extensive as that of the right side, a portion of the bony wall being retained to establish its relation to the optic nerve.

Head IV (Fig. 5).—Only the ethmoid cells have been prepared with their mucosa intact. Usual relation.

Head V (Fig. 6).—Small opening in the sphenoid roof is seen and also in the last posterior ethmoid cells, the mucosa of the external wall of the left side being retained. The usual relation of the optic nerve and last posterior ethmoid cell is definitely shown.

Head VI (Fig. 7).—In this, the relation of the optic chiasm to the two sphenoids is well shown and also the usual relation of the nerves to the right and left last posterior ethmoid cells.

Head VII (Fig. 8).—The left sphenoid is observed in relation with the chiasm and both optic nerves. The mucosa of the right sphenoid is seen projecting upwards into the last posterior ethmoid cells on that side, being separated from the optic nerve by the wall and a portion of the cavity of the ethmoid cell. The right optic nerve courses along the external wall of this cell for at least 1.5 centimeters, corresponding to the observation made by Loeb.

With this confirmation increased emphasis is given to the claim made that the optic nerve becomes more vulnerable to infection of sinus origin whenever through replacement of the sphenoid cavity it runs in close proximity to the outer wall of the last ethmoid cell.

Designating one the angular relation and the unusual form, the mural relation, the results of the stuly of the twenty-three heads are as follows:

| | Angular | Per Cent | Mural | Per Cent |
|---|---|---|---|---|
| Loeb's series | 28 | 93.3 | 2 | 6.7 |
| Present series | 15 | 93.8 | 1 | 6.2 |
| Total | 43 | 93.5 | 3 | 6.5 |

The remarkable agreement of these findings suggests further investigation along these lines.

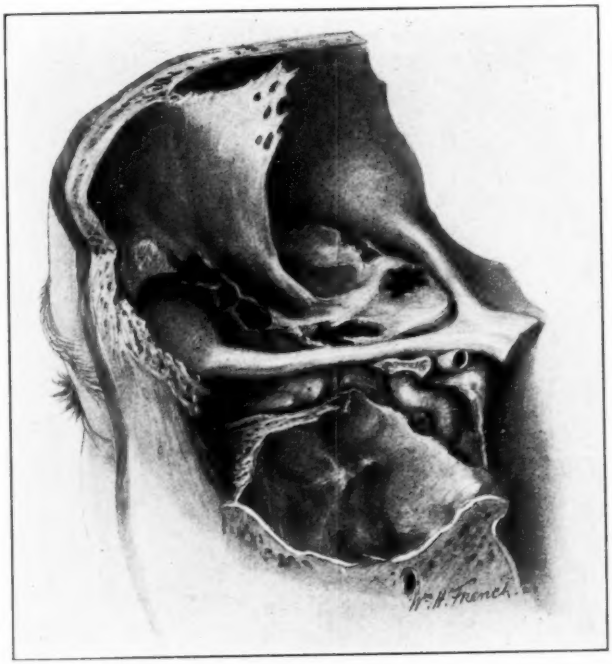
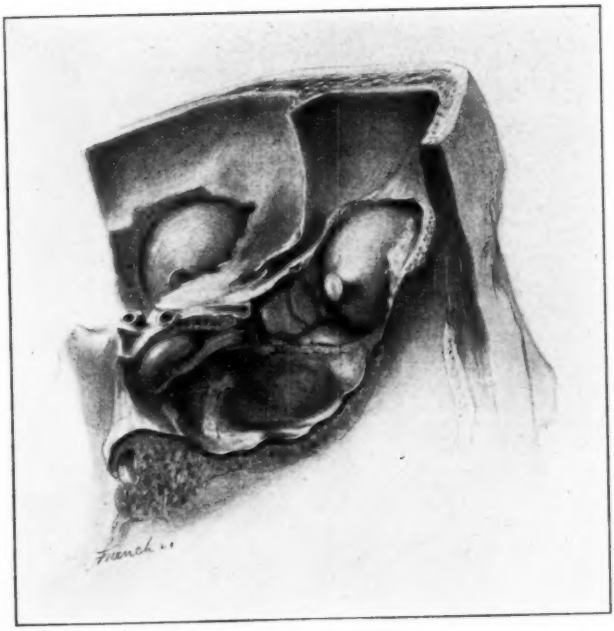



Figure 1.



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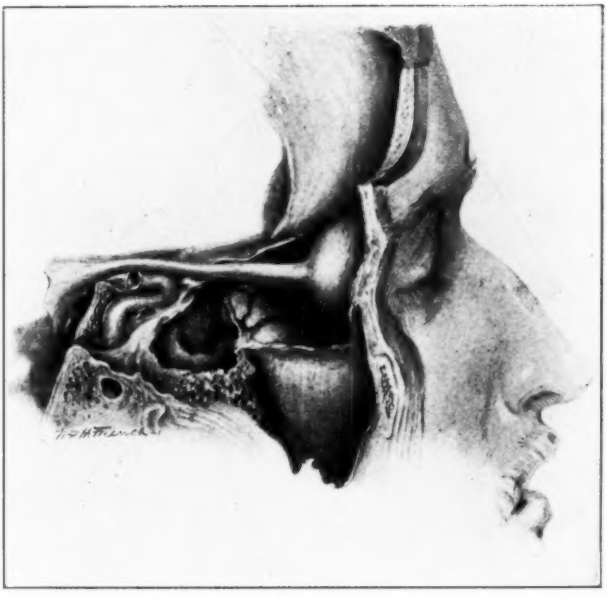

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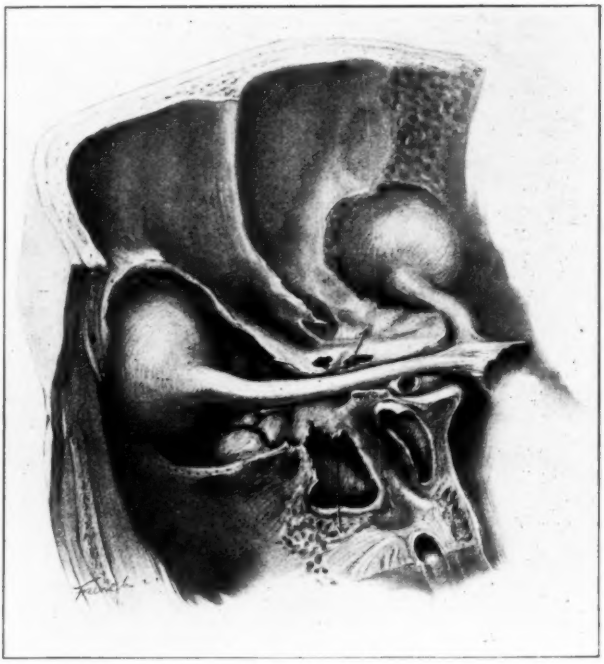

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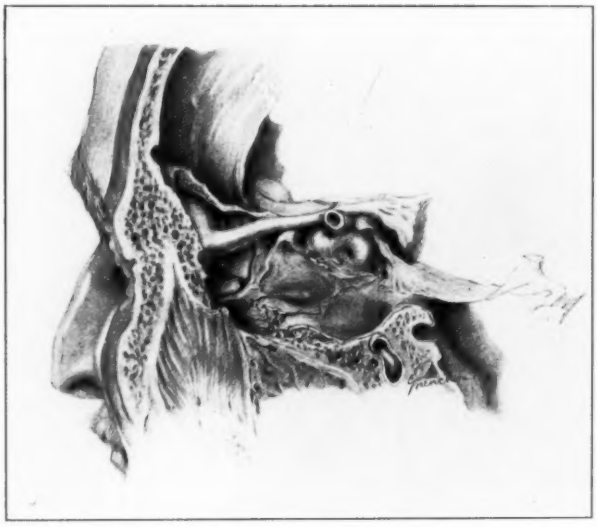

Figure 5.

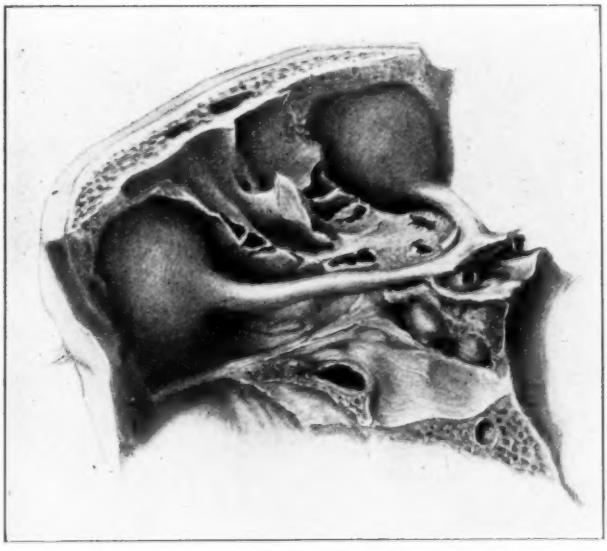

Figure 6.

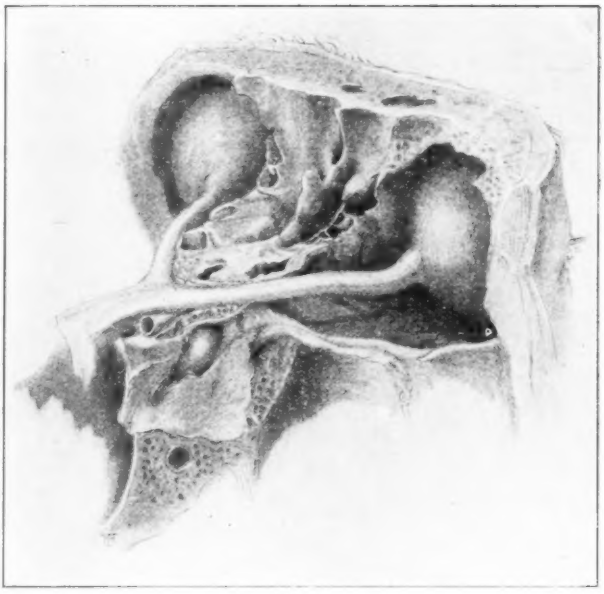

Figure 7.

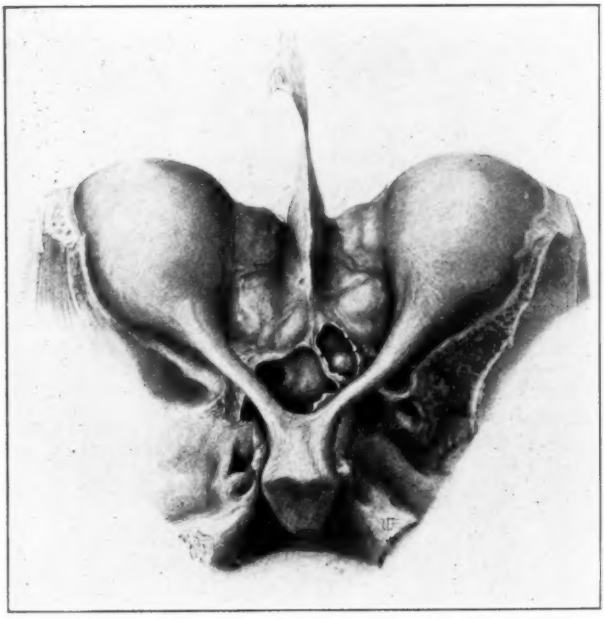

Figure 8.

LII.

# A STUDY OF THE MECHANISM OF PAIN AS SEEN IN RHINOLOGIC AND LARYNGOLOGIC CASES.

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Few problems in rhinology and laryngology are more difficult and involved than those centering about the interpretation of pain. The purpose of this paper is to explain briefly its mechanism. In order to understand this properly, it is well to classify pain into three general types—first, somatic; second, autonomic, and third, psychic pain, as the mode of production of these three types differs.

## MECHANISM OF SOMATIC PAIN.

Somatic pain is due to irritation of the sensory nerve endings (pain sense organs) and of the pain nerve fibers. The latter are afferent somatic neurons, which have their cell bodies in the sensory, posterior root ganglia and pass into the central nervous system by the posterior nerve roots. The pain impulses, transmitted along the afferent somatic neurons and the pain tract within the central nervous system, finally reach the optic thalamus (containing the pain center) and the cerebrum. The latter becomes conscious of the stimulus and recognizes the place where it arose.

## MECHANISM OF AUTONOMIC OR VISCERAL PAIN.

Autonomic pain is due to irritation of afferent autonomic nerve fibers of the viscera or of the structures that are visceral in origin—i. e., those that are developed embryologically from the branchial arches and pouches. These afferent autonomic nerve fibers have their cell bodies, like the afferent somatic fibers, in the posterior root ganglia (somatic sensory).[1] The pain impulses are, therefore, transmitted to some of these

sensory ganglionic cells and pain arises. The cerebrum recognizes this pain and forms a judgment of its locality. That locality, however, is not the region where the pain stimulus arose, but in the peripheral distribution of the somatic sensory nerves, whose cells in the posterior root ganglia had been stimulated. This is why autonomic pain is also called referred pain.

### MECHANISM OF PSYCHIC PAIN.

Psychic pain originates in the cerebrum of neurotic persons and from there is projected to the periphery. The location and the character of this pain are influenced by the ideas the patients have about it. It is described as dull, sharp, constant or "coming and going," burning, twisting, scratching, etc.

Neurotic persons do not distinguish between discomfort and pain. Their nervous system seems to be so sensitized that the slightest discomfort makes them complain of severe pain.

The most important characteristics of psychic pain are the facts that it usually does not interfere with sleep and that superficial contacts, like combing the hair, are very painful, while deeper pressure may cause no discomfort.

### PAIN OF NASAL ORIGIN.

The interpretation of pain as a result of nasal disorders is rather difficult. It is due to the complexity of the nasal innervation. The nose is not only supplied by a nerve of special sense, but also by somatic sensory and most probably by afferent autonomic nerves. It may, therefore, be considered not only as an organ of special sense but also as a somatic and visceral organ. We know how difficult it is at times to interpret pain caused by somatic sensory disturbances alone. The difficulty is still greater when we try to interpret pain caused by autonomic or visceral disturbances alone. The nose is an organ in which disease may produce both types of pain, either singly or combined. Besides, many neurotic patients suffering from psychic pain may show nasal abnormalities which are not the cause of the pain. Therefore, in our specialty, we may encounter the three types of pain previously mentioned, namely: (1) Somatic, (2) autonomic, (3) psychic. Before discussing in what manner the various nasal diseases may cause

pain it is well to describe the nerve supply of the nose in a little more detail than is usually done.

NERVE OF SPECIAL SENSATION.

The nerve of the special sense of smell is the olfactory or first cranial nerve. It has nothing to do with pain. Irritation of this nerve, however, may produce a disagreeable as well as an agreeable sensation. I know of a very sensitive patient who complains of getting a "headache" whenever she smells certain odors.

SOMATIC SENSORY INNERVATION OF THE NOSE.

The first and second division of the fifth cranial nerve constitute the somatic sensory innervation of the nose. According to those modern anatomists and physiologists[2] who have given this subject their attention, the sphenopalatine ganglion does not supply the nose with sensation, as is stated in all textbooks of rhinology. The socalled sensory branches of this ganglion are continuations of the branches of the second division of the fifth cranial nerve. Bearing this in mind, let us restate the sensory supply of the nose.

OUTER NOSE.

Side of the nose, upper part; infratrochlear branch of the nasociliary nerve of the first division of the fifth cranial nerve. Side of the nose, lower part; nasal nerve of the infraorbital nerve of the second division. Tip and bridge of the nose; external nasal branch of the nasociliary nerve of the first division.

INNERVATION OF THE NASAL CAVITIES.

Septum, anterior part: anterior ethmoidal nerve (internal branch of nasociliary nerve); middle part: nasopalatine nerve of second division; posterior part: posterior superior nasal branches of the second division.

Outer wall, in front of the turbinates: anterior ethmoidal nerve (external branch of nasociliary).

Superior turbinate, superior meatus, middle turbinate: posterior superior nasal branches of the second division.

Middle meatus, inferior turbinate, inferior meatus: inferior nasal branch of large or anterior palatine nerve of second division. The anterior part of the inferior meatus receives

also a branch of the anterior superior dental nerve of the second division.

### THE ACCESSORY SINUSES.

Antrum of Highmore: branches of the superior dental nerves of the second division.

Frontal and anterior ethmoidal sinuses: fibers of the nasociliary of the first division have been traced by Meckel and Langenbeck.[3]

Posterior ethmoidal and sphenoidal sinuses: posterior superior nasal branches of the second division; a sphenoethmoidal branch of the nasociliary nerve has been described by Luschka, which passes through the posterior ethmoidal foramen and is distributed to the sphenoidal sinus and the posterior ethmoidal cells in front of the body of the sphenoid; orbital branches of the second division, according to Luschka, supply, besides the periosteum of the orbit, also the periosteum and mucous membrane of the posterior ethmoidal cells and sphenoid sinus.

### NASAL SOMATIC PAIN.

Nasal somatic pain may be caused by disorders affecting (a) the peripheral nerve endings (pain sense organs); (b) the trunks of any of the above mentioned nerves. Let us trace how the pain impulse from the nose reaches consciousness. It starts in the peripheral nerve endings (pain sense organs), which end "free" between the epithelial cells of the mucous membrane[4] of the nose and accessory sinuses. It may also begin in the nerve fibers themselves. It passes along these fibers to the Gasserian ganglion, where the cell bodies of all the somatic sensory nerves of the nose are located. From these cells central processes are given off which enter the pons and bifurcate into ascending and descending branches (roots). They end in the sensory nucleus of the fifth nerve. Here the neurons of the first order in the sensory pathway from the nose to the cerebrum stop and those of the second order begin. The latter pass from the cells of the sensory nucleus to the upper part of the pons, where most of them cross to the opposite side and form the trigeminothalamic tract. They end in a special portion of the optic thalamus, which is considered to be the

center of organic pain. From here impulses are transmitted over neurons of the third order to the sensory area of the cerebral cortex.

### NASAL AUTONOMIC PAIN.

The nose, as mentioned above, can also be considered as a visceral organ. Like other such organs it is supplied by both the sympathetic and the parasympathetic nervous system. It is known that these systems furnish the nose with vasomotor and secretory motor nerves.[5] The important question: "Do afferent autonomic fibers exist in this locality?" as in the thoracicolumbar region, has not yet been definitely decided. There is clinical evidence, however, that such may be the case. There are cases on record where the Gasserian ganglion had been extirpated or the sensory root of the fifth nerve avulsed and yet pain was still present. Also pain referred to regions far removed from the distribution of the fifth cranial nerve occurs, and its explanation must be sought in a disturbance of afferent autonomic fibers. There is another related fact that would make us surmise that such fibers may be present within the nose. It is known that in the thoracic and lumbar region the white communicating rami contain afferent autonomic fibers which pass to the somatic sensory ganglia, where they have their cell bodies. The vidian nerve has also been considered as a white communicating ramus, which is formed by the large superficial petrosal nerve. It may, therefore, be assumed that it also contains afferent autonomic fibers. If it is true that afferent autonomic fibers are present within the nose, their probable course would be as follows: Accompanying the intranasal branches of the second division of the fifth cranial nerve, they pass through the sphenopalatine ganglion to the vidian nerve, and have their cell bodies probably in the geniculate ganglion, and possibly also in some of the cervical and upper thoracic ganglia. The geniculate ganglion gives off a branch to the middle ear, and so we can explain the occurrence of otalgia of nasal origin. From the cervical and upper thoracic ganglia fibers are given off which make up the sensory supply of the mastoid region, the occiput, the neck, the shoulder, the arm, the forearm, the hands and the fingers. Thus we are able to explain the pain in these regions as a re-

sult of irritation of the afferent autonomic fibers within the nose.

Let us trace now how an autonomic pain impulse from the nasal cavity reaches consciousness. It arises in the afferent autonomic nerve fibers, if it is true that such fibers are present within the nose. It passes along these fibers, which have their course within the nasal sensory branches of the second division of the fifth cranial nerve, through the sphenopalatine ganglion to the vidian nerve. From here it is transmitted through the large superficial petrosal nerve to the geniculate ganglion, and probably through the carotid plexus and the communicating rami to the cervical and upper thoracic ganglia. From these sensory ganglia it travels along the somatic sensory pathway to the optic thalamus and cerebrum. When it reaches the cerebrum the pain is referred along the peripheral distribution of those somatic sensory nerves whose cells in the geniculate, cervical and upper thoracic ganglia had been stimulated by it.

It is obvious that we cannot have nasal autonomic pain unless afferent autonomic fibers are present within the nose. It is also obvious that these fibers can be irritated in any part of their course, near the sphenopalatine ganglion or away from it. The term "sphenopalatine ganglion neurosis," as used by Sluder, would make one believe that the irritation of these afferent autonomic fibers can only occur at this ganglion, which I think we have no reason to assume, or that the pain is due to irritation of the ganglion itself. The fact that the application of cocain to the region of the sphenopalatine ganglion relieves all or most of the pains may be due to the circumstance that near this region we can get at practically the entire sensory innervation of the nose which is blocked by the cocain. It is also possible that this drug may anesthetize the afferent autonomic fibers, although, as far as I know, its effect on such fibers has never been demonstrated. The sphenopalatine ganglion, being an autonomic ganglion, has nothing to do with pain, except possibly with the causation of pain in the throat. Winkler[6] is the only neuroanatomist of note who believes that this ganglion contains a few sensory cells, which give off fibers to the soft palate but none to the nose. This is why it is possible to have pain in the throat in disease around the sphenopalatine ganglion.

What is the difference, if any, between autonomic and somatic pain? It is obviously difficult to determine whether the pain impulses from the nose reach the central nervous system by way of the afferent somatic or the afferent autonomic neurons, as the peripheral processes of the latter use the former as a pathway. It is difficult to conceive that the afferent autonomic fibers are irritated without simultaneous irritation of the afferent somatic fibers, unless we assume that the irritant is of such a nature that it would affect one set of fibers only, which may be possible. It is most probable, however, that both systems of nerves may become involved and that most of the pains are due to involvement of the somatic sensory system. It is impossible to distinguish from the character of the pain whether it is of autonomic or somatic origin. But when the pain is referred to regions far removed from the distribution of the terminal filaments of the fifth cranial nerve it may be considered as referred pain of autonomic origin. Associated symptoms, like sudden pallor or sudden flushing, salivations, etc., may also help us in deciding whether the pain is of autonomic origin or not.

NASAL DISEASES THAT CAUSE PAIN.

1. Suppurative Diseases of the Outer Nose.—Among the suppurative diseases of the outer nose that cause severe pain the following are the most frequent: Furunculosis, folliculitis, cellulitis and erysipelas. A folliculitis is quite frequently overlooked. It is necessary to inspect carefully the entrance of the nose to find the lesion. The pain in these diseases is due to irritation of the nerve endings or nerve fibers of either the nasal branch of the infraorbital nerve of the second division or of the external nasal and infratrochlear branches of the nasociliary of the first division, depending upon the location of the lesions.

2.—Septal Abscess.—A septal abscess is frequently accompanied by severe pain. It is due to irritation of the nerve endings or nerve filaments of the anterior ethmoidal nerve, a branch of the nasociliary nerve.

3. Pressure of Turbinates on Septum.—Many observers have described pain due to contact between the septum and the turbinates. It is supposed that in these cases inflammatory

conditions in the nasal sinuses are absent and that relief is obtained by shrinking the mucous membrane of the turbinates and the septum in contact with it, by means of cocain or adrenalin, or by operation. However, the frequent occurrences of anatomic variations in the nasal cavities, producing contact points between the turbinates and the septum, without pain, makes one doubt whether it is ever caused in this way. It is most probably due to an obscure nasal sinusitis, which is the cause of the congested and enlarged turbinates.

4. Vacuum Pain.—Vacuum pain is supposed to be due to nondiseased and obstructed sinuses. Readers are referred to Sluder's work for the explanation of its mechanism.[7]

5. Acute Nasal Sinusitis.—Affections of the accessory sinuses of the nose are the disorders par excellence that produce pain. In the acute cases there are two forms of pain: first, a constant pain, localized more or less over the diseased sinus, and second, a paroxysmal pain, neuralgic in character.

The constant pain over the sinus can be explained by the involvement of the very sensitive periosteum of its wall. It is also possible that at the same time with the inflammatory changes of the mucous membrane of the sinus its sensory nerve filaments also suffer inflammatory changes. In the latter instance a neuritis is probably present. It is known that in neuritis the pain is usually constant, and its location corresponds to the distribution of the nerve involved. Therefore, a knowledge of the sensory innervation of the diseased sinus will help us to a certain extent in the diagnosis. The paroxysmal pain, however, does not by its location indicate the involvement of a particular sinus. This pain is most probably caused by a neuralgia of the irritated nerve fibers of the diseased sinus. In any neuralgic condition we know that the pain does not correspond to the distribution of the particular nerve involved, but it appears in certain rather ill defined zones and areas corresponding roughly to the different trunks of the main nerve.

### THE TIME OF APPEARANCE OF THE PAROXYSMAL PAIN.

The paroxysmal pain, which may result from the involvement of any of the nasal sinuses, is characterized by its appearance at a certain definite hour of the day. The opinion

that it is due to periodic increased secretion of pus is not based on accurate observation. A sinus may be full of pus without causing any pain. Of course, this does not mean that an empyema of a nasal sinus does not produce pain but only that it does not explain its periodic appearance. It is possible, however, that the habits of the particular patient have something to do with it. It is known that certain factors which increase blood pressure, like smoking, alcohol, stooping, also increase the pain. It is, moreover, recognized that the blood pressure undergoes variations during the twenty-four hours of the day. Faught[8] calls attention to the ever-varying conditions, both internal and external to the body, that are continually at work and result in a varying blood pressure picture. Janeway[9] has reported daily systolic variations amounting to 60 mm. of mercury. We may, therefore, assume that changes in the circulation of the blood which increase blood pressure have an influence on the appearance of the pain.

When a patient gives a history of pain occurring at a particular time, we should inquire as to his habits of life, his routine of work, how and when he eats. The blood pressure should be taken at frequent intervals during the day.

6. Chornic Nasal Sinusitis.—In chronic sinusitis pain is usually absent. If present, it is frequently characterized by a diffuse headache, which is probably due to toxic absorption from the infected sinus or sinuses. Patients suffering from chronic sinusitis are also subject to acute exacerbations, during which time the pain assumes the character of an acute sinusitis. There are patients with chronic sinusitis that suffer very severe pain, even without acute attacks. It is supposed to be due to an osteitis of the nasal sinuses, which causes pressure on the sensory nerve filaments. There are undoubtedly patients, however, who have a complete osteitis of the ethmoid and sphenoid sinuses, as may be demonstrated during an operation, and yet they do not complain of any pain. There must, therefore, be some other factors besides the osteitis that would explain this severe pain. It is possible that it is due to a disease of the nerve fibers themselves. In order to demonstrate this it would be necessary to make careful sections of the operated material from the nasal sinuses and prepare these sections with the special

stains for nervous tissue. This is a very tedious and difficult procedure and pathologists are not anxious to do it for us.

It is important to keep in mind that patients suffering from chronic sinusitis without pain may develop this symptom subsequent to an operation on the sinuses. This should be a warning to those who consider minor and annoying nasal symptoms as sufficient indications for this very serious operation.

### CONSIDERATION OF THE SEPARATE SINUSES.

Acute Maxillary Sinusitis.—In acute maxillary sinusitis the pain may be either constant and localized over the antrum (periosteal and neuritic), or neuralgic in character, affecting the terminal filaments of the posterior superior, middle superior and anterior superior dental nerves of the second division of the fifth cranial nerve. The pain radiates, therefore, to the incisor, canine, bicuspid and molar teeth, also along the infraorbital nerve to the lower eyelid, side of the nose, cheek and upper lip.

Chronic Maxillary Sinusitis.—In chronic maxillary sinusitis the pain may be not only in the region of the antrum and reflected along the terminal nerve filaments of the second division of the fifth nerve, but also in the frontal region, even if the frontal sinus is absolutely normal or absent. To explain this phenomenon is not easy. As a matter of fact, involvement of any of the nasal sinuses may cause frontal pain. The probable reason for this occurrence is the fact that an irritation in any branch of the sensory distribution of the fifth cranial nerve may be the exciting cause of reflex pain in any other branch of the same nerve. As the frontal nerve is clinically known to be one of the most sensitive nerves of the body, an irritation in any part of the fifth nerve is easily reflected to it. Pain of this character is called by some referred pain. It is well, however, to call it reflex or reflected pain and to limit the term referred pain to autonomic pain.

Acute Frontal Sinusitis.—In acute frontal sinusitis the pain is either (a) constant with a sense of fullness and tension over the frontal region (periosteal pain) or (b) neuralgic in character, coming on in paroxysms, over the frontal region and radiating to the anterior temporal or parietal region. It

is due to disease or irritation of the terminal filaments of the nasociliary nerve of the first division.

Chronic Frontal Sinusitis.—In chronic frontal sinusitis the pain is usually not so severe and not so constant as in the acute form.

Acute Anterior Ethmoiditis.—In acute ethmoiditis the pain is either constant over the bridge of the nose or projected in paroxysms over the frontal and vertical region. It is due to involvement of the anterior ethmoid nerve of the nasociliary of the first division.

Chronic Anterior Ethmoiditis.—In chronic ethmoiditis the pain is of the same character but less marked. It may be absent.

Acute Posterior Ethmoiditis and Sphenoiditis.—In acute posterior ethmoiditis and sphenoiditis the pain may be (a) frontal, constant, due probably to disease of the sphenoethmoid branch of the nasociliary of the first division, or paroxysmal, due to irritation of the same branch. It may, however, be reflex in origin; (b) retroorbital, more or less constant, due probably to involvement of the orbital branches of the second division, which supply the periosteum of the sphenoidal and posterior ethmoid cells as well as the periosteum of the orbit; (c) occipital, due probably to involvement of the afferent autonomic fibers.

Sphenoid sinusitis may cause neuralgic pains along any of three divisions of the fifth cranial nerve. This, according to Sluder,[10] is due to the close relationship of the sphenoid sinus to the Gasserian ganglion. This would also explain the appearance of herpes during an attack of sphenoid sinusitis.

Chronic Posterior Ethmoiditis and Sphenoiditis.—In chronic posterior ethmoiditis and sphenoiditis the pain is similar in character, but less constant than in the acute forms of the disease. There are patients, however, with chronic ethmoiditis and sphenoiditis, who suffer the most agonizing pains imaginable.

7. Malignant Tumors of the Nose.—In malignant tumors of the nose pain is a prominent symptom. The tumor may cause pressure on the nerve endings or nerve fibers, and the site of the tumor will to a certain extent determine the location of the pain.

Pain in these cases may also be due to blood and lymph stasis. The tumor compresses the vessels of the nasal mucous membrane, which are in communication with the veins of the dura and the superior longitudinal sinus. The blood and the lymph stasis of the nose, causing a stasis in the dura mater of the brain, will produce pain.

8. Relation Between the Nasal Sinuses and the Meninges of the Brain.—It is well known that the nasal sinuses are anatomically closely related to the frontal cerebral lobes and the base of the brain. By means of the vascular and the lymph systems a communication between the nasal cavity and the meninges is brought about. The only meningeal membrane that is sensitive to pain is the dura mater. It is supplied in its anterior and middle portion by sensory terminal filaments of the fifth cranial nerve, and in its posterior portion by a branch from the sensory ganglion of the root of the vagus nerve.

Toxic absorption from the nasal sinuses seem to cause pain, which is not confined to the area of a particular nerve but is diffuse. It is most probably caused by irritation of the meningeal branches of the fifth and tenth cranial nerves and is usually accompanied by mental depression, inability to concentrate and loss of memory.

## TRIFACIAL NEURALGIA.

In the preceding paragraphs pains due to nasal disease affecting (a) the nerve endings, (b) the nerve trunks, and possibly (c) the Gasserian ganglion were considered. They were described as constant and localized, or paroxysmal and neuralgic. The paroxysmal pain is an example of what is usually referred to as symptomatic neuralgia. It has absolutely no relation to what is called trifacial neuralgia or tic douloureaux. In these cases the source of the pain is not to be found in the tissues of the organ from which it is transmitted to the sensorium through the sensory nerve (trifacial or fifth) supplying the tissues, but rather that it is due to some action on the sensory trunk itself.

The etiology of this terrible disease is obscure. Histologic investigations do not enlighten us fully. There is probably a biochemical change in the nerve, of whose nature we know

nothing. It is, therefore, necessary to keep in mind that a diagnosis of trifacial neuralgia is a diagnosis by exclusion.

REPORT OF A CASE.

A large number of cases could be cited that have been studied by me from the standpoint of the presenting symptom —pain—but space does not permit. I want, however, to report very briefly a type of case that we meet so frequently in practice.

A neurotic woman, 30 years of age, married, Hebrew, well nourished, came to the Manhattan Eye, Ear and Throat Hospital in December, 1921, complaining for two years of constant and severe pain over the frontal, vertical and occipital regions. She had been in this country only two years. She came from Hungary, where she suffered greatly during the world war. The pain she complained of was present only during the waking hours, never at night when sleeping. Stroking of the hair with a comb was very painful. She was examined by several physicians, who apparently did not find any organic trouble, and she was referred to the hospital for examination of the nasal sinuses. Anterior rhinoscopy showed a high deviation of the nasal septum, with pressure on the middle turbinates, and there was some mucopus between the latter and the outer nasal wall. Posterior rhinoscopy, pharyngeal and laryngeal examinations were negative. X-ray report by Dr. Law, December 31, 1921: "Left frontal shows mild involvement. Right frontal shows moderate involvement. Ethmoids show moderate involvement with considerable absorption of the posterior septa. Both antra show mild involvement. Sphenoids large and clear."

To give the patient the benefit of the doubt, a submucous resection of the nasal septum was done. The anterior tips of both middle turbinates were removed. The Loré suction irrigation apparatus was then used for six months. The pain has not been relieved. It is still present only during the waking hours and never interferes with sleep. The patient looks well and is gaining weight. She weeps and cries on the slightest provocation. Nasal examination at present is absolutely negative. Another X-ray examination was made on August 22, 1922, and Dr. Law reports all sinuses practically clear.

This demonstrates the efficacy of the nasal treatment, which cleared up the sinuses but not the pain.

Comment: This patient undoubtedly had a chronic sinusitis but her pains were not caused by it. Whenever a healthy looking and neurotic patient complains of constant pain, day by day, which does not interfere with sleep, its most probable origin is a neurosis. Before doing any operative work on the nose it should be remembered that psychic pain may on superficial examination simulate that due to nasal sinusitis, especially if certain signs of nasal disease or anatomic variations are present.

### PHARYNX AND LARYNX.

It is impossible in a communication of this character to discuss all diseases of the pharynx and larynx that may cause pain. Only some phases of the subject which are of interest to the laryngologist will be considered.

### HERPES OF THE PHARYNX.

Herpes of the pharynx is caused by an inflammatory condition of the sensory ganglion of the ninth nerve. It is usually not recognized. There is severe pain on swallowing, and a sticking pain in the throat even without swallowing. The pain may be so severe that patients will refuse food. Otalgia is also present. On examination we find vesicles surrounded by a red areola on the base of the tongue, on the palate or on the pharyngeal wall.

### TUMORS OF THE NASOPHARYNX.

Malignant tumors of the nasopharynx may involve the Gasserian ganglion. It is important to remember that these tumors may not cause subjective symptoms until the skull is penetrated. Shelden[11] of the Mayo clinic has observed sixty-nine tumors of the nasopharynx, nine of which have been attended with intracranial extensions. Of these nine tumors, four have exhibited the Gasserian ganglion syndrome. Pain is usually the initial symptom and the one for which relief is sought.

### LARYNX.

Pain is present in all acute pus infections of the larynx. Its intensity depends on the location of the lesion. In infections

of the epiglottis and the arytenoepiglottidean folds the pain is most severe, because this area has a very rich sensory distribution, and also there is here more irritation on swallowing. In herpes of this area the pain is so severe that it is impossible to take any nourishment. It is due to an inflammatory process of the sensory ganglion of the vagus nerve. It may, therefore, be accompanied by other vagal symptoms, like frequent vomiting, slow and irregular pulse and otalgia.

In chronic diseases of the larynx, like tubercular ulcers and extralaryngeal carcinoma, pain is a prominent symptom. In tuberculosis of the larynx the pain may be so severe that it is justifiable to inject the superior laryngeal nerve with alcohol at its place of entrance through the thyrohyoid membrane. In carcinoma of the larynx the pain depends upon the location of the tumor. Extralaryngeal tumors give rise very early to pain on swallowing, which may be the first presenting symptom. We should not forget, however, that lesions in this area may cause reflex otalgia. I know of a patient who came to the Lenox Hill Dispensary complaining only of pain in the right ear. Examination of the ear was negative, but an involvement of the right half of the larynx was found. Microscopic examination confirmed the diagnosis of extralaryngeal carcinoma of the larynx.

In tabes dorsalis laryngeal pain, of a burning and lightning nature, is a prominent symptom in the socalled laryngeal crisis.

### THYROIDITIS.

In thyroiditis the thyroid gland, as a whole or in part, becomes swollen and tender. There is pain on swallowing. The pain may radiate to the region back of the ear. Two such cases have been seen by me at the Manhattan Eye, Ear and Throat Hospital. They both complained of pain on swallowing and of a choking sensation. The thyroiditis in both cases was preceded by an acute tonsillitis. When seen at the hospital no acute inflammatory condition was evident in the throat.

### ANGINA PECTORIS.

Occasionally pain in the throat and teeth are the only manifestations of an angina pectoris. The diagnosis is made on

the evidence of arteriosclerosis, pain on pressure of the carotid arteries and typical anginoid attacks in the interval.

### GLOBUS HYSTERICUS.

Globus hystericus is a painful construction in the throat, due to a hyperesthesia of the esophageal and pharyngeal mucous membrane, causing a contraction of the musculature of the esophagus and pharynx. The diagnosis of this condition is easy if the patient has the classical feeling of a globe rising from the cardiac end of the stomach behind the sternum to the throat, where the globe gets "stuck," and if other hysterical symptoms are present. But when the patient complains of a painful feeling of constriction alone in the throat we should be careful not to call it a globus hystericus. We are more liable to make the mistake if the patient is a more or less nervous woman. We should remember that it may be the only symptom of an angina pectoris. Another cause of a feeling of a constriction in the throat may be a heart insufficiency, which produces a congestion and a sensation as if the blood rushes to the throat. Hyperthyroidism may produce such a feeling with a sensation of choking. It is also very frequently caused by flatulence, after a large meal, especially where there is a slight cardiac insufficiency.

Direct irritation of the pharyngeal branches of the vagus nerve which supply the muscles of the pharynx may produce a contraction of these muscles and a painful feeling of constriction. This irritation may be brought about by mediastinal disease, by intrathoracic goiter, by aneurysm of aorta, etc.

An example of a possible hyperirritability of the pharyngeal branches of the vagus nerve without discoverable cause was recently seen at the Manhattan Eye, Ear and Throat Hospital. A perfectly healthy young man came to the hospital complaining of a choking sensation and a painful feeling of constriction in the throat. Examination of the throat was absolutely negative. Atropin sulphate, which has a paralyzing effect on the vagus nerve, was given in gr. 1/100 doses, twice a day. After taking a few doses of the atropin the symptoms were gone. Occasional treatment with the same drug prevents the reappearance of the symptoms.

### HEADACHE (DIFFUSED PAINS).

The limitations of this paper forbid a complete discussion of headache. A few points, however, of interest to the laryngologist will be emphasized. We ought to be able to distinguish between pains of the head and headache. Pain, being due to irritation of the nerve endings or nerve fibers, will be more or less localized. Headache, on the other hand, being due to toxic absorption from the diseased tissues and to an involvement of the dura mater is diffuse. Pain, therefore, is a local symptom, and headache a focal symptom. The focus of infection may be in head, neck or in some other part of the body.

It is known that there is a more or less intimate connection between the vagus centers and the fifth cranial nerve. Thus Head[12] has described certain cephalic zones which he considers particularly involved and painful in the presence of disorders of the thoracic and abdominal viscera, which are supplied by the vagus nerve. These zones are at present of not much value to us. It should be remembered, however, that a good dose of castor oil will sometimes relieve a headache that is supposed to be due to nasal disease.

### INDURATIVE OR RHEUMATIC HEADACHE.

Another type of focal headache is the one usually referred to as indurative or rheumatic headache. It is characterized by fibrous deposits or nodes at the insertions of the scalp and neck muscles. It occurs mostly in the occipital region and is worse on motion and in damp and windy weather. A characteristic symptom of these patients is the dizziness they complain of, especially on bending down. It is most probably due to a disturbance of the sensory nerves of the involved muscles. Normally these nerves of deep sensibility transmit to the central nervous system sensations of position and movement of the head and neck. In disease of the muscles implicating these nerves the central nervous system receives false impressions on movement of the head and neck which leads to dizziness.

### MIGRAINE.

Migraine is an hereditary and obscure type of headache. It is a periodic neurosis in which there is a nervous discharge

affecting not only the fifth but also other cranial nerves as well as the autonomic nervous system. It is a general disease in which the headache is only one symptom. There are many theories to explain its causation. The one most accepted by neurologists is that it is due to a vasomotor disturbance, which causes an intracranial vasoconstriction and an anemia of the brain. If we accept this theory, the question still remains: "How is the pain produced?" That a portion of the brain, for instance the sensory nucleus of the fifth nerve, can be irritated by an anemia does not appear rational. According to Lewandowsky,[13] the pain is possibly due to the contraction of the walls of the blood vessels of the brain. Nasal disturbances have also been thought to be causative factors.

HEADACHE IN ENCEPHALITIS.

It should be remembered that violent headache may not only accompany but also be one of the sequelæ of epidemic encephalitis. It may occur months after the process has subsided. If this is not kept in mind mistakes in the diagnosis are easily made.[14]

PITUITARY HEADACHE.

Pituitary headache is due to a disproportion between the sella turcica and the pituitary gland, producing pressure on the sensory nerves of the dura mater. The pain is usually bitemporal or deep in the forehead behind the eyes. It is relieved by the administration of the whole pituitary gland in about one grain doses, three times a day. The following case may serve as an example:

A man, 32 years of age, bookkeeper, came to the Lenox Hill Dispensary, on August 28, 1922, complaining of pain deep in the forehead behind the eyes. He suffered this pain for the last four years. It always appears after exertion, like going upstairs, or after any kind of exercise. His eyes were examined by several ophthalmologists, who prescribed eyeglasses. The pain, however, was not relieved. He used to take a great deal of aspirin, which weakened his heart but had very little effect on the pain. Before he came to the dispensary he was treated for "heart trouble" in one of the leading hospitals of New York. There he was advised to have his nose examined. It was thought that his pain was due to a nasal sinusitis.

Examination showed that the nose and throat were absolutely negative. X-ray report, August 30, 1922, Lenox Hill Hospital: The frontals, ethmoids and antra do not reveal any pathology. The sphenoid sinuses show cloudiness at their posterior part. The sella is small, the posterior and anterior clinoids appear to meet. The dorsum sellæ is irregular and thinned out.

The patient was given pituitary extract, grain 1, three times a day. After a week's treatment the pain practically disappeared and has not returned up to the present time (December, 1922).

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LIII.

# RECENT NASAL FRACTURES.

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It is to be regretted that current literature so rarely brings to the mind of the student and general practitioner the pressing need of proper care and attention to fractures of the nose. It is the universal opinion of those experienced in this work that there are few cases of fractures in any location on the skeleton that require more constant care and vigilance than fractures of the ossa nasi; and it may be added that there is no facial organ more sensitive to deformity in its relation to disfigurement of the face.

Far more was written on this subject in the uncertain past than appears in the periodicals of the present day. The older recommendations consisted principally of enthusiastic descriptions of forceps, splints and complicated mechanical devices calculated to hold the set nose securely in place until union resulted, apparatus which at the present time is no longer heard of, or which was even disavowed later by the authors themselves. Very few of the procedures in the long history of the subject survive, not through wrong effort or lack of ingenious design, but rather because the various mechanisms submitted were built to meet individual case requirements, hypothetical cases or artificial lesions produced on the cadaver. The error was for the most part due to an effort to treat all fractures of the nose by a single method. Considering the great varieties of nasal fractures, due to varying degrees of force applied in varying directions by objects of all shapes and sizes at different levels of the nasal bridge, it is readily conceivable that the number of lesions to be produced is infinite, and it is evident that no single method or immobilizing device can be applicable to such a multiform lesion. The older practitioners would seem to have failed to realize that probably no man sees two cases of fracture of the nose exactly alike.

II.

Injuries of the nose may be divided into two primary classes —injuries without loss of tissue, and trauma with destruction of bone, cartilage and soft structures; and as a result of the varying degree of force in varying directions the resulting injury ranges from minor dislocations of the columnar cartilages to complete crushing injuries of the nasal arch and septum.

The supporting structures of the nose, whether osseous or cartilaginous, are subject to three classes of trauma—i. e., dislocation, infracture and complete fracture, and all cases of injury will fall under one or more of these classes. The involvement of the septum is, however, usually limited to dislocation and fracture.

Injury to the septum in a large proportion of cases is of course due to its anatomic position as a rigid central support for the bridge of the nose, and from its exposed position the quadrilateral cartilage suffers more frequently, though to no greater degree than the osseous portion. Zuckerkandl, whose figures are widely quoted in this connection, found that in 80 per cent of the skulls examined the bony septum (perpendicular plate of the ethmoid) extended as far down as the middle of the long axis of the nasal bones or even lower, and in only 3 per cent did not reach to the upper margin of the ossa nasi.

Practically all fractures of the nose are of the compound type, and this point must be kept in mind during the entire course of treatment, even though, as occasionally happens, there is little or no loss of blood from the nares to indicate laceration of the mucous membrane. It is common practice, due doubtless to the infrequency of complications, to disregard this factor, which is unsurgical to say the least, and which has led to serious and even fatal complications. There is not, it is true, the same degree of serious significance attached to these compound fractures as attends those of other parts of the body, immunity being granted by several probable factors. The injured bone is ordinarily thin and shell like, consequently the exposed broken edges are very thin and are readily sealed by the coagulation of blood coming from the minute sinuses

and capillaries of the injured and exposed surface. The marked vascularity of the supporting and lining structures of the nose doubtlessly confers a local immunity to infection during exposures of the tissues, and finally we may consider that because bacteriologic examinations of the normal mucous membrane show attenuated strains of bacteria present in a locus theoretically believed to hold a special immunity to those germs normally present, danger is remote except in the presence of complicating infections, such as rhinitis and sinus disease.

III.

The size, form and general appearance of the injured feature may be greatly altered by swelling, ecchymosis and the bruising of surrounding soft tissue, the reaction being so great that the detection of fracture and dislocation is extremely difficult and uncertain. When the swelling has at least partially subsided, or in those cases where the reaction to injury of the soft parts has been less severe, it is possible to obtain an exact idea of the extent and degree of injury by external and intranasal examination. The deformity presented by simple inspection often reveals the nature of the injury without palpation or radiograph, but the importance of careful intranasal inspection cannot be overemphasized. The septum may be dislocated and so much displaced laterally as to considerably narrow or even completely occlude one nostril without altering the external contour of the nose.

Dislocation.—The most frequent cartilage dislocation is quite naturally a separation of the quadrilateral cartilage at its junction with the perpendicular plate and with the vomer. This results usually in an apparent and actual depression of the flexible portion of the external bridge, with the tip of the nose frequently directed to the side of the dislocation. If the septum, as a result of direct violence, is dislocated backwards the resulting deformity is fairly typical; a deep depression immediately beyond the lower extremities of the nasal bones and a slight but unnatural tilting up of the tip of the nose. Both of these types of dislocation are accompanied by partial or complete separation of the cartilage from the nasal process of the superior maxilla, complete separation being responsible

for the frequently observed widening of the columella due to encroachment of the anterior angle of the cartilage into the nostril.

Infractures.—Infractures of the cartilaginous or osseous structures of the nose offer perhaps the most annoying difficulties of diagnosis, and it is in the majority of these cases that unsatisfactory end results are obtained. With crepitus absent and no help lent by the Roentgen ray, and with all acute swelling apparently subsided, the perichondritis or periostitis which may persist for weeks disguises a cosmetic irregularity in a manner likely to indicate a satisfactory prognosis which may later be modified by cruel fact. Tactile examination must indeed be highly specialized to determine depressions of infractures hidden by firm indurated periostitic swelling.

Fractures.—The concept regarding the osseous structure of the nose as an architecturally supported arch is apparent in the writings of earlier surgeons and is sometimes met with in the present day. That the nasal projection is a poor substitute for an arch from the engineering standpoint is readily conceded by those who have frequently attempted its reconstruction.

The extreme rarity of nasal fractures compared to injuries of other bones of the skeleton must not be ascribed to any special resistance of the socalled arch itself, but rather to the infrequency with which the organ is subject to injury. It is well to remember in considering this point that the same remarkable automatic protection of the eyes by instinct—i. e., the jerking of the head or the raising of the arms to ward off impending blows, does more to protect the nasal bridge than does the structure of the bridge itself. It is a fact borne out by a study of comparative anatomy that the skulls of mammals of the lower orders show a far greater support of the nasal arch and bridge (up to and including the negroid skull) than is present in the Caucasian race.

Statistics on fractures in general from the central European medical centers show nasal fractures to have a frequency of from 1 to 1.6 per cent. According to the few available hospital reports in this country, these figures seem high, due either to the fewer number of adherents to dangerous sports or to the

fact that many of these cases are considered ambulatory and are privately treated in physicians' offices.

In practically all traumatizations of the nose with external direct force to the osseous bridge there is a tendency of the nasal bones to separate from each other, as there is practically no obliteration of the central suture in the white race. Carter states that bony union is frequently seen in the negro and Mongolian races, and that there is constant osseous union in apes. This tendency to separation is responsible for the great frequency of dislocation of the nasal bones. It is doubtlessly true that the nasal bones are more frequently dislocated from the frontal processes of the superior maxillæ or separated from each other than actually fractured.

The most common injury then involves the separation of the nasal bones and their fracture or both. This condition presents marked deformity depending on the force applied. There is an overriding of the nasal bones on the nasal processes, a flattened deformity of the bridge resulting. Marked flattening is accompanied by backward dislocation of both nasal bones and septum, and may be of sufficient degree to produce an undue prominence of the nasal processes of the superior maxillæ, causing an apparent widening of the disance between he eyes.

With a forcible blow striking the nose from the side, one or both nasal bones may be fractured and displaced from their beds and the organ is pushed to one side. In blows from the same direction but applied with lesser force only the nasal bone of that side is broken with a depressed deformity. Laterally applied blows to the bridge of the nose in children may produce a depressed deformity due to fracture on the same side and a greenstick or infracture of the opposite nasal bone.

The chief lines of fracture traverse the lower half of the nasal bones in a more or less oblique direction; the ossa nasi are thinnest at the lower extremity, requiring only a light blow properly applied to cause a separation of continuity. The fracture is frequently seen to traverse the bone at a point corresponding to the nasal frontal spine. Occasionally the break may be seen to run longitudinally following the grain of the bone, especially when the force has been applied with a small object.

Fractures of the septum may occur independently of fractures of the nasal bridge, and, as Jaravay pointed out years ago, innumerable fractures of the cartilaginous septum occur without any injury to bone, and with so little discomfort or reaction the lesion may remain unknown to the possessor.

The most usual location of fractures of the septum is in the posterior two-thirds of the cartilaginous portion and the anterior half of the osseous portion, the latter usually in the perpendicular plate, the vomer being protected by virtue of its deep position.

IV.

Hippocrates thought that union of the fragments in nose fractures was generally complete in six days. Boyer, Malgaigne and other early writers believed that these fractures healed with great rapidity. Molinie stated that eight days as a minimum is necessary for fixation. It is rather conceded by contemporary writers that fixed union does not occur till late in the second week or well into the third.

Correction of old septal deformities disclose a difference in the method of natural repair of fractures of bone and cartilage. Osseous repair or union is made by means of hyperplastic callus and new bone formation. Repair of cartilaginous fractures is by simple connective tissue callus.

V.

The complications resulting from fractures of the nose are many and varied. Probably the most ordinary complication in practice is a septal hematoma, which may lead to abscess formation. Low lateral injury may cause laceration of the lacrimal duct. The line of fracture may extend upwards into the frontal bone and frontal sinus, causing an emphysema when attempt is made to blow the nose. The same type of injury in greater degree may cause the line of fracture to reach the anterior fossa with accompanying symptoms of cerebral concussion and other intracranial injuries. Rogers has reported three cases of fracture of the cribriform plate and death from meningitis. Severe crushing comminuted fractures have involved the maxillary sinus and even anterior ethmoid cells. A late complication is chronic suppurative periostitis or perichondritis, recognized by fetid odor, purulent discharge and

pain. In these cases the condition persists, and the nose remains sensitive until necrotic bone or cartilage is discharged by nature or removed surgically. Of the rarer local complications cavernous sinus thrombosis and ophthalmoplegia (Dowling) may be mentioned.

VI.

Treatment of nasal fractures is best considered in two divisions: (a) reduction and (b) retention. The question of when to undertake these measures is still open to discussion. Garel, Harold, Foster, Lee Cohen and others insist on an immediate institution of treatment, while many operators feel that a more thorough diagnosis and a more exact approximation of the separated parts can be made if the circulatory reaction following the blow has subsided. I have personally set no hard and fast rule to cover the question and allow myself to be governed by the individual case. A fair proportion of these injuries show surprisingly little swelling and edema, and in many the swelling from the circulatory disturbance is soft enough to permit thorough tactile examination. When the reaction is severe enough to prevent a complete study of the supporting structures I allow a period extending up to ninety-six hours in which to allay the reaction by the use of ice packs, rest in bed and elevation of the head. At the end of this period I feel certain enough of the condition of the nose to perform reduction.

(a)

Almost every known instrument with proper slenderness and rigidity has been used and recommended for the intranasal reduction of fractures. Septal periosteotomes, female silver catheters, Volkmann's curette, the Tripier dilator, artery snaps, the Martin forceps come to mind as recommended by various authors. A number of special forceps for reducing the septum have been devised, of which the most popular is doubtless the Adams instrument, especially as modified by Carter.

The rules governing the reduction of nasal fractures vary almost as greatly as do the types of injuries. Simple reduction by traction with subsequent natural retention, as in fractures of the long bones, is possible in so few of the cases that

the percentage is practically negligible. Reduction is usually a matter of reconstruction of the superstructure of the nose with immediate support, which must be maintained until rigidity is established. Dr. Lee Cohen believes that this can be accomplished with best end results by thoroughly mobilizing the nose with the Adams-Carter forceps, eliminating all resistance of the injured or uninjured supporting structures, so that when placed in a splint the nose may be molded into proper form.

(b)

For intranasal retention of the reduced fracture a large variety of methods have been used. From the earliest days of lint packing, splints of various design and material have been conceived. Rubber tubing, cups of rubber and gutta percha, hollow tubular splints of rubber and metal have been hailed with enthusiasm and then forgotten. Probably the gauze pack and the Simpson sponge are the only examples of this type of retention apparatus which we use today.

Of the mechanical devices few have been of practical value. Probably the most successful of these is the apparatus conceived by Martin and Martin; a four pad mechanism arranged winglike, which is introduced into the nostril closed. A plate resting on the floor of each nostril acts as an anchor, and the turning of a key outside of the nose opens the wings of each side simultaneously so that one pad exerts pressure against the lateral wall of the nose while another applies itself against the septum wall. Molliere, Chandeloux and more recently Nelaton, Ombredanne and others, find that the apparatus is seldom tolerated by the nasal mucous membrane, though the Martins maintain that the nose can sustain the apparatus for long periods of time with proper regulation of tension. Variations of this apparatus are used by the Martins, Sargnon and others for the correction of recent fractures and deformities.

Molinie devised an internal expanding splint which is now recommended chiefly for the treatment of old fractures. The opened splint gives no lateral pressure, but being triangular in form and resting on the floor of the nose with its base, it exerts tension along the entire line of the bridge close to the median line.

The external splints are as varied and almost as complicated in some instances as the internal splints. Molliere introduced his external plaster method in 1888. Prior to this time other external apparatus had been devised, such as the lead gutter of Malgaigne, molds made of rubber (Hamilton), papier mache (Dzondi), dachylum (Weber), collodium (Dumreicher) and dental plate composition (Garel).

Warbasse recommends as recently as 1918 a U-shaped rigid appliance attached to a headband descending from the forehead to enclose the nose, from the side of which padded thumbscrews are applied to exert pressure at desired points. This apparatus is further improved by a flat metallic tongue attached to the bottom of the U so that it lies over the upper lip. This permits varying degrees of counter pressure by applying a bandage around the head above the pinnæ and beneath the nostrils.

In this country the apparatus of Carter, devised as an external support to the nasal bridge, was at one time popular. This consisted of a rigid adjustable arch set over the nose, its base resting on the frontal processes of the superior maxillæ. Splints of gutta percha were inserted and adjusted in the nostrils with thread and needles attached, which were passed through the tissues of the nose and secured externally to the rigid arch. Frequent loosening of the tension and bathing of the parts are said to have prevented necrosis.

A modification of this splint was suggested by Grant in 1903. This writer omitted the use of the internal splints and passed silver wire through the depressed deformity, attaching it to a steel arch straddling the bridge, the arch resting on rubber cheek plates retained by means of tapes passing around the head.

Other external mechanical splints have met with more success. When used in selected cases the device of Adams has been very satisfactory. It consists of two lateral pads fastened by rigid swivels to a firm head band and adjusted against the side of the nose by a thumbscrew. This is similar to the adjustable pads of Joseph of Berlin. Gamgee's apparatus presses on the side of the nose by means of a screw and spring, with lateral movement of the apparatus prevented by means of a spectacle frame hooked to the ears. Cobb later devised a

similar appliance, held in place by a stiff metal head band fitted carefully to the circumference of the head.

The ingenious device of Von Eck deserves mention. It consists of a rigid metal saddle held over the bridge of the nose by buckled tapes around the head. Through this saddle are inserted six adjustable thumbscrews, set in various positions, which press on a flexible metal plate in direct contact with the nose. By lowering or raising the several screws the inner plate, which is padded, can be made to conform to the requirements of many individual cases.

Most rhinologists have a collection of many of these devices locked away in cabinets reserved for tonsillotomes. It is wrong to condemn them all as useless, for many of them are applicable to selected cases; the principal objection is the extreme rarity in which a given apparatus may be properly applied.

The sheet copper splint, which Cohen attributes to Roe, but which practically is identical with the zinc splint of Daly, is proving to be the best retention apparatus we have at our command. To properly construct the splint, which is made to fit each individual case, a paper pattern saddle is cut to fit the injured nose, and the resulting truncated triangle is used to cut a similar shape from a sheet of 24 gauge sheet copper with tinner's shears. The edges of the copper are smoothed with a file and the entire surface and edges are covered with a layer of adhesive plaster and an inner lining of soft thin cotton.

### TECHNIC.

Dr. Cohen believes, and I am inclined to believe that most of us with experience in this work agree with him, that it is most essential in the reduction of all fractures to first thoroughly mobilize the entire bony framework of the nose, so that when placed in the splint it may be molded into proper form. Unless this is done reduction cannot be satisfactorily accomplished, and even if the reduction seems to be successful there is always a tendency of the nose to assume a faulty position, which grows more manifest as the swelling disappears.

To produce mobilization (I am quoting Lee Cohen at length) the nasal bone is grasped with the Carter-Adams forceps, one blade of which is introducted into the vestibule of the nose and the other (covered with rubber tubing to prevent injury to the skin) is placed on the outside. The bone is then twisted with sufficient force to assure complete mobilization, and the same process is repeated on the other side.

Following this the same forceps are reintroduced, both blades bare, into the nostrils, one blade on each side of the septum, so that, by a lifting motion or twisting, the nasal bones can be brought forward and upward into their proper relation with the nasal process of the maxillary bone below and the frontal bone above. This manipulation serves at the same time to bring the septum of the nose into proper alignment.

To retain the desired position the nasal chambers are lightly packed with plain pulled gauze in continuous strips one-half inch wide, care being taken to elevate with firm but gentle pressure those portions showing a tendency to depression, in certain marked tendencies an overcorrection being advisable. The light resting of the thumb and index finger externally with one hand during the packing serves to guide the operator's judgment.

The copper splint just described is then placed on the nose and is held in place by an inch wide strip of adhesive tape extending across the nose and face from beneath one ear to the other. A small strip of adhesive plaster extending from the forehead down to the lower end of the splint, reinforced by two cross strips on the forehead, prevents any tendency of the splint to slide downward and exert undue pressure on the tip of the nose.

It is important to remember that great care must be taken to insure an equal tension to the cheek strips holding the splint in place, as an uneven pull will easily cause the axis of the nose to get out of alignment.

The first dressing is left for four days, and then changed every third day. If, however, there appears to have been any displacement of internal packing or external splint during daily observation, it must of course be redressed at once. The

packing may be discontinued after ten to fourteen days. The splint should be used from three to four weeks.

In conclusion let three elementary principles of general surgery be recalled in the treatment of these cases: First, asepsis, especially in the compound fractures; second, suture of lacerated skin with a view of minimizing scar formation, and third, the use of tetanus antitoxin when indicated by the nature of the injury.

LIV.

# THE OTOLOGIST IN THE FIELD OF SOCIOLOGY (THE ALLIANCE OF MEDICAL AND SOCIAL SCIENCES IN THE PREVENTION AND PALLIATION OF ACQUIRED DEAFNESS).*

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Luke, the beloved physician, records an incident in the life of the Nazarene when a certain lawyer sought a reply to the question "Who is my neighbor?" His reply was as follows: "A certain man went down from Jerusalem to Jericho, and fell among thieves, which stripped him of his raiment, and wounded him, and departed, leaving him half dead. And by chance there came down a certain priest that way; and when he saw him, he passed by on the other side. And likewise a Levite, when he was at the place, came and looked on him, and passed by on the other side. But a certain Samaritan, as he journeyed, came where he was; and when he saw him, he had compassion on him. And went to him, and bound up his wounds, pouring in oil and wine, and set him on his own beast, and brought him to an inn, and took care of him. And on the morrow, when he departed, he took out two pence, and gave them to the host, and said unto him, Take care of him; and whatsoever thou spendest more, when I come again, I will repay thee. Which now of these three, thinkest thou, was neighbor unto him that fell among the thieves?" The lawyer answered: "He that shewed mercy on him." Then said the Nazarene: "Go, and do thou likewise."

To the physician disciple this parable must have made a strong appeal, for to the physician, not only on account of the nature of his avocation and the Hippocratic oath to which he has subscribed, but because of the human element in his experience and training, responsibility as neighbor to his pa-

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tient becomes imperative. The peculiar psychology surrounding his deafened patient vastly enhances this responsibility. Every handicap in human experience has its psychologic bearings; every calamity in life its woes; but none except obvious fatal diseases provokes more absolute helplessness and depression than defective hearing.

In the presence of this audience of otologists I need not attempt to enlarge upon this phase of the hearing problem. I know of no more trying experience than to be called upon by a deafened patient to give a final opinion, when no hope of permanent improvement may be held out. We older otologists frequently are obliged to face this ordeal. In the functioning of this experience the prevention of deafness plays no part, for in every instance the days of prevention have passed. Likewise in the majority of cases no well founded hope for improvement can conscientiously be vouchsafed. We must face the bald facts surrounding his deafened condition, not only in the patient's interest, but it must be in a manner that will preserve our own integrity and honesty of purpose. At this very point arises the otologist's opportunity to become his patient's neighbor in the highest sense, and it is here that he may profitably enter the field of sociology, at least so far as sociology as a science may pertain to the welfare of his deafened patient. It is one thing for the otologist, after having conducted all the hearing tests, to conscientiusly tell a deafened person, even in the kindest way, that his deafness must remain permanent. It is quite another attainment to go to the necessary length of demonstrating to him how to overcome his handicap. The first is an ordinary professional business transaction—the second requires time, self-sacrifice and toil beyond the realm of financial remuneration. The otologist who contents himself with the rendering of his scientific opinion only, must be classed with the priest or the Levite who came and looked on and passed by on the other side. The citation of a few experiences will serve to illustrate:

A boy of fourteen summers whose mother stated to a social worker that she had formerly consulted a number of otologists, who all agreed that her son's deafness was incurable, but they failed to tell her what could be done to lessen the handicap. At last she consulted another otologist (not the writer), who

is deeply interested in organization work for the deafened and who is constantly in touch with its workers. He told her that her son must study lip reading without delay; that his vocation must be carefully selected and his whole trend of life guided in the direction of an avocation which he could fill with satisfaction to himself and to his friends. He was placed in a private school of lip reading. Later he entered a high school, taking a cooperative industrial course, and finally landed a remunerative job. His mother's gratitude overflowed her vocabulary—her problem was solved at last. "That—that —is a hero man" was her cry.

A young woman about twenty-two years of age consulted one of the most distinguished members of this society. His attitude was friendly, his examination thorough, every test complete and painstaking, leaving nothing unaccomplished that could add to the thoroughness or the value of his scientific opinion. Upon completion of the examination he told her plainly that her deafness was permanent and that no form of treatment held out any hope for permanent amelioration. She paid his fee and went home to spend the days of agony, hopelessness and despondency of those who must remain in the "silent land." She saw no ray of hope, either in the way of occupation, of social enjoyment or of education—the whole world was dark. Finally, some friend persuaded her to consult another otologist, who arrived at exactly the same hearing results, but who devoted the required time in an endeavor to open the door of hope. He explained to her that her defect was her peculiar handicap, that people all over the world with few exceptions were victims of one handicap or another. He explained the peculiar psychosis of deafness; how it grappled with one's optimism; he related incidents where people far worse than she had come into lives of comparative happiness and usefulness; he urged her in the strongest terms to rebuild her life on the basis of her handicap by making a careful study of lip reading, by using artificial aids for hearing, by associating herself with other deafened people in the field of social service work and by preparing for remunerative employment. She was introduced to the social service workers in the New York League for the Hard of Hearing and entered actively into its work. She was told that while no direct

benefit might come from constant treatment, there were times while suffering from severe colds that a moderate amount of nasal and aural treatment would be beneficial. It was impressed upon her that she could remain under the advice and counsel of a competent otologist who would be primarily her guide and counselor regarding all things pertaining to the hearing function. She responded to these suggestions with eagerness. This young woman has completely adjusted herself to her handicap and is happy. She is following a useful and remunerative occupation. She is becoming a competent lip reader and, strange to say, actually thinks she hears better than formerly.

One of my own experiences was a woman of about thirty-three years of age from a distant city, who had gradually from her twentieth year grown deaf from otosclerosis. I was called upon to say the final word and could not conscientiously evade the responsibility. She was despondent, rebellious, belligerent and refused to be comforted. She would not even consent to investigate the various artificial aids for hearing. I finally obtained her consent to go with me and confer with some people who are much deafer. I called at her hotel and took her to our League headquarters, where we spent an hour in a conference with our deafened workers, all of whom are happy and contented. We persuaded her that the wearing of a hearing device tended to make friends and to retain them, for the reason that one's friends are glad to talk into the hearing device, knowing that no special effort is required. She was shown through the various departments; educational, vocational, occupational, etc. She was urged to make a study of lip reading and to learn the things that would be of the greatest benefit in overcoming the handicap of deafness, and finally she succumbed to the influence.

Hundreds of cases of this kind might be cited through various organizations of the hard of hearing throughout the land. In another communication* I cited the case of a boy who had gradually grown deaf from the age of thirteen until twenty. When he graduated from high school he refused to enter college, stating as his reason that he did not like to

*Volta Review, November, 1921.

impart to his mother that he was afraid to go through the same deafened experience in college as in high school. It was at this time that I advised him to join our League for the hard of hearing and take advantage of its various departmental work, particularly lip reading. In a personal communication received from him he relates his League experiences. His letter says: "I have started one of the strangest transformations in my life. I have begun to look at the world in a different light. You advised me to join the lip reading class as soon as possible, and I attended one at the League rooms. I will never forget my first night of lip reading. I came full of hope, for I had been told that lip reading was a wonderful thing and expected to learn the entire art in one lesson. How my hopes were dashed to pieces. All around people were nodding their heads in the negative or affirmative as the case may be; at times they would burst out in laughter at something the teacher had said. I sat there all alone watching them and cursing myself—what mockery, what foolishness. I almost cried, but as time went on, my point of view gradually changed—I found out it wasn't so bad, after all. That a person who was deafened did not have to isolate himself from the world. At the present time I have accomplished a great deal in lip reading. I also come into contact with other activities of the club, especially its social life. I have taken part in three or four amateur dramatic performances. I joined the dancing class; in fact, my entire life has been changed by this experience. Sometimes I pinch myself to see if it is all real. Words cannot express the work that the young women of the League are performing. Their cheerfulness and happiness take one out of the deepest depths of gloom and misery." This young man is now in his senior year in a dental school and is accomplishing his work with very little difficulty.

The above remarks give emphasis to the fact that the otologist's responsibility does not end with the medical care of his deafened patient. He must carefully weigh every factor that will tend, first, to the elimination of his hearing defect, providing such relief is possible, but in the more serious cases he should assume the attitude of the Samaritan in the parable and become the neighbor, the teacher, adviser and guide of his

deafened patient. He should be prepared to educate the public, particularly along the lines of preventive deafness; to recommend the prophylaxis in individual cases, and he should encourage all organized efforts to ameliorate the unhappy condition of deafened people. In short, the otologist, together with the social worker in charge of the necessary activities for the deafened, must impart the necessary courage, information and hope to enable him to rebuild his life upon the new basis. "In the metamorphosis from the morbidity and shrinking self-pity to happiness and usefulness" more or less character rebuilding and readjustment become necessary.

### SENSIBLE SUGGESTIONS FROM THE DEAFENED INDIVIDUAL'S STANDPOINT.

Mr. John de Raismes Storey, in an article on the "Psychology of Deafened People,"* emphasizes the necessity of overcoming the obstacles within as well as those which are objective. Deafened himself, he refuses to be a typically deafened man and endeavors to be successfully deaf. To use his own words: "By that I mean he must strive to live a moral, wholesome life with an occupation suited to his capacity, with friends, hobbies, wide interests, etc., and not to let his affliction affect his character except beneficially." "I do not feel that the deafened man should accept a humble task if he is fitted by attainments for a better one." These are the sentiments of a deafened man who has achieved success in the legal profession and who is in active practice today.

At the third annual meeting of the American Federation of Organizations for the Hard of Hearing, held in Toledo, June, 1922, interesting papers were presented by deafened people representing a diversity of occupations. "The Deafened Man in Business Life" was discussed by Mr. Starling W. Childs of New York City, who is a member of a prominent brokerage house. When Dr. Phillips asked me to come here he gave me as a subject, "The Deafened Man in Business,"† and I took it. He meant me to discuss this from my own experience rather than to treat it along more theoretical

*Volta Review, May, 1920.
†Volta Review, Nov., 1922.

lines. I have done this, and you will understand that what I have to say today I have tested and found true by the best of tests—day by day experience. And by the same token, you will also understand that I am not dealing with the man who is totally deaf, but rather with the question of how people who suffer from various degrees of impaired hearing may be expected to get along in the world of affairs. Persons who are hard of hearing hold within themselves the key to their own destiny. If they allow themselves to be weak, discouraged and despondent they will for a time receive the sympathy they obviously seek, but if they persist the chances are that they will first be pitied, then endured, and then avoided. "Instead of weakly deploring our hard lot, we should strike a balance (as the business man would say), wherein our assets, as well as our liabilities are listed, and by steadily conserving the former and improving them we can be reasonably sure of some day showing a balance on the right side of the ledger." We are often bored by friends with discussions of their general ailments, and in a way they seem proud of them, but the deafened man—almost never. This probably arises from the fact that deafness is not an obvious defect, but the attitude is a wholly mistaken one and is sure to be a great obstacle to his success. Instead of concealment and vain subterfuge, his attitude should be one of complete and unembarrassed frankness. By frankly admitting his difficulty he will at once put those about him at their ease, and I can say with absolute assurance that he will almost invariably find his fellow men helpful and sympathetically cooperative. And do not forget that where real study of a subject or piece of business is required the deaf man has one very positive advantage. By shutting off his hearing apparatus he is at once free of all the disconcerting and disturbing noises which constantly assail the ears of his associates, and is thus enabled to concentrate absolutely upon the problem before him. In short, the deafened man will find by experience that his balance sheet has assets as well as liabilities. He then commends the employment of hearing devices and concludes with the statement that the deafened man is better equipped for some kinds of work, particularly in accounting and other business which require great concentration (In short, Mr. Childs has shown

his power of concentration by winning several matches of golf from the writer of this paper.)

"The Deafened Woman in Society" was a paper presented by Mrs. George L. McAlpin,* who stated that even if modesty causes one to shrink from relating personal experiences, for the cause of humanity and for the sake of service one would not remain silent. To sympathize, to help, to encourage, these must be common experiences; and with the deafened I feel sure that the suffering and the struggle are the same, no matter what the environment. One day a beautiful star appeared on my horizon. Faintly it gleamed at first, often obscured by the fleeting clouds of despair. That star was the birth of hope. Was it possible that my eyes could be trained to take the place of my ears? Was it possible that I could keep in touch with my family and friends in delightful intercourse? It was possible. I would try. All of this happened some years ago, but the star is still shining, leading me to new worlds to conquer and new lessons to learn. From the first I sought speech reading. Three things I count necessary to success along social lines: Courage, culture and a determined effort to master the art of speech reading. Limitations must be met and recognized, but I have yet to meet the situation where a courageous and frank attitude will not attract companionship. There is always a kind friend who will give a clue to the subject of conversation. Culture is an ornament that is especially becoming to those who have lost their hearing. A hobby is an excellent thing for the deafened. A determined effort to master speech reading is essential to all success. It is said that there are good speech readers and poor speech readers. I say we can all be good speech readers if we will persevere. And one of the most important things to do is to cultivate and develop the faculty of observation.

"The Deafened Woman in Professional Life"† was a paper read by Miss Julia E. Johnsen. She stated that the deafened woman in professional life assumes there is a handicap. This is only relatively true. It is true that we do not like, on account of our deafness, to approach people, to give them special

*Volta Review, November, 1922.
†Volta Review, November, 1922.

trouble, to place ourselves in a conspicuous or embarrassing position. Our sensitiveness or pride gets to the front. This is psychologic and not a handicap unless we make it so. We must not confuse any disadvantage with the fact of deafness, except as deafness contributes to the state of mind. What we need is clear thought and sensible conclusion that we will do whatever comes to us to do. Wrong mental attitudes are not held by us alone. Most obstacles can be overcome. We do not expect that deafened physicians, nurses, lawyers, and musicians, for example, can do exactly the same things as one who hears. Such training, or a strong attraction for special work, is too valuable not to be made use of. One might, for example, work with an assistant; or a physician might specialize in laboratory or X-ray work; a lawyer might turn from court to take up brief and record work, research, law librarianship, or even cataloging of legal literature. A musician might, like Beethoven, turn to composing, or to harmony or song writing; because music is not only a matter of physical sound, it is a matter of internal sound as well. I mention these things because one who has started out toward a professional career is apt to turn aside when deafness comes upon him, a fact which he may afterwards regret. In the case of a reporter, the ability to write well can be turned to authorship. We have a reporter who has become very successful in magazine writing. There are business standards to be met, rules, methods, resources or working facts to be learned. When these are learned the way is comparatively easy—that is to say, the work is related to one's capacity for the task in hand, and I think deafness is very little hindrance. This has been my own experience. My own work, however, is especially adapted, because it calls for very little communication. It is editorial work of a special kind and also reference library work. For the sake of the future it is better to expect too much of the deafened than to do the harm and lack the vision of not expecting enough.

A paper on "Compensations" was presented by Mrs. Robert C. Morris,* who said in part that he who has been afflicted with deafness only a short time is apt to be rebellious, and he

*Volta Review, December, 1922.

is skeptical that this universal law of compensation can possibly apply to his case. But he who has been deaf from infancy, through childhood and youth, and through middle age, knows that there is some soul of goodness in things evil. He knows that, as Emerson said, he can, like the wounded oyster, mend his shell with a pearl. When one of our five senses has become impaired the four others become strengthened beyond the normal. Intuition develops rapidly if we educate ourselves to know those flashes of knowledge which come to us intuitively, and the deafened may have through the life which must be lived in contact with other people, a more dependable guide than persons with normal hearing. The deaf person naturally becomes an accurate reader of human nature. They rarely make mistakes in judging men and women. He watches the lips of any new acquaintance, knowing that the eyes can be trained to deceive and other features may wear a mask, but the mouth is the only feature that cannot be made to camouflage the truth. Thus the deaf person selects his friends with infallible intuition. He sees in people important things that we people never see at all. His faculty for observation becomes the foundation for memory, reason, judgment and all kinds of culture. The deaf person not only has time to think, but he learns to concentrate. If he cannot carry on a conversation with John Jones or Wilbur Smith, it is just as well, perhaps, for the John Joneses and the Wilbur Smiths of the world seldom say anything that is worth listening to. If the sphere of the friendships of the deaf person is circumscribed, he may have quality if not quantity, and if he cannot become acquainted with a wide circle of friends, then he may turn his attention to Shakespeare and Emerson and Stevenson, friends who unite the whole world. If we cannot hear a lecture we may console ourselves with this thought: that the best of lectures always find their way into print, and 90 per cent of the lectures, so far as literary values are concerned, are not fit for print. Most of us can build a world of joy and accomplishments with such tools as we have. To be able to hear as the average person does is just to be an average person, but to be able to do something—to specialize—to do something better than the average person can do it, is to cause a thousand people who can hear to seek the path to our door. The

hard of hearing person is sympathetic, and the world needs that kind of understanding sympathy. Shakespeare said: "None can be called deformed but the unkind." We have come from many circles, we represent social and professional relationships of many kinds, and in this convention, perhaps for the very first time in our lives, we are among our very own people, for we are the people who understand each other so much better. Only you and I know the world in which we live, and I am proud to be numbered among your gallant company of the deafened. This very experience is one of the great compensations that deafness has brought to me.

These are but a few of the papers which have recently been written by highly intelligent people along the same lines, but time will only permit a few quotations from a remarkable article "On the Technic of Being Deaf," written by Earnest Elmo Calkins, a prominent figure in the literary and advertising world, and published in the January number of the Atlantic Monthly. It is unfortunate that we deafened can go so long without detection. We frequently choose foolishly, preferring to pass as slow, thick-headed, stupid persons rather than as the quick-witted deaf persons we really are. I have become a master of the art of being deaf. It is an acquired art. People are no more born with it than they are born bachelors. A bachelor is something more than a man who has failed of marrying; and the art of being deaf is something more than loss of hearing. When I asked Mr. Edison why he of all persons did not avail himself of one of the electric devices for making hearing less difficult, he said: "Too busy. A lot of time is wasted in listening. If I had one of those things my wife would want to talk to me all the time." Discussing a banquet at which it might be possible for me to be "seen," I said to a friend: "I'm not going to any more of those dinners. It's such a bore to sit there and not hear any of the speeches." "Not so much of a bore as if you heard them," was his feeling reply. After all, are not the deaf rather inclined to overrate the mere accomplishment of hearing? Audition is not without its drawbacks, and deafness without its compensations. For us, the noisy world is soft pedaled. My deafness beginning at fourteen years of age, my teacher, a woman of rare sympathy and helpfulness, would not permit me to fail and

insisted upon helping a discouraged boy to keep up with his classes. I still remember the enthusiasm with which she once said: "Earnest, I want you to succeed, not in spite of your deafness, but on account of it." There ought to be a book of short biographies of the famous deaf, telling how they met their problem. Such men as E. S. Martin, Thomas Edison, Sir Joshua Reynolds, Ludwig von Beethoven, the Earl of Chesterfield, Harriet Martineau, etc. We cannot draw any consolation from the belief that our condition is tragic. To begin with the first lesson and the hardest, it is imperative to admit that one is deaf—admit to oneself, and tell the world, and accept the penalties as well as the compensations. As some cornfed philosopher remarked, "Old maids is really the happiest—after they quits strugglin'." After all, the deaf are not deaf all the time; for they are not deaf when there is no occasion to hear. The deaf person should always prepare in advance for as many emergencies as possible. He should study the time table in advance, read the signs on the walls and in the train-shed. I have learned to set myself for awakening at any morning hour. Nothing can compare with a gainful occupation. He then mentions aids to hearing, lip reading, which is little short of marvelous, and then classifies the liabilities and assets in the way of recreation which a deafened individual may well commit to memory. On my index expurgatorious are:

Conversation in the best sense; the theater; lectures; public dinners and most private ones; music; social dancing; games like "What is my thought like?"; being read aloud to.

I have left: Books; pictures, moving and stationary; art, painting, sculpture, architecture and applied natural science; scenery; travel, on foot, train, boat, horse and motor; exhibition dancing, and all spectacles and pageants; games like golf and whist; nearly all hobbies.

Someone has said (boy, page Mr. Bartlett) that when God closes one window, He opens another a little wider. I have tried to help Him and swing my window altogether open. Among his recreations which he calls startling are painting, modeling, carving, fishing, digging in the earth, and play games. In consequence I have never been bored. I make models of ancient ships, which gives an excuse for hunting

old books and prints to learn how they looked and were rigged. I have a colony of bees, which are sufficiently amiable to permit me to take out the brood combs and find the queen, when there is someone to see me show off and exclaim: "Don't they ever sting you?" I also battle with beetles and worms, for the satisfaction of raising a few of the varieties of roses. If I play a rotten game of golf, it is not because I am deaf. There is no reason why a deaf man should not be a very good golfer. With me it is, perhaps, because I enjoy a walk as much with a blackthorn stick in my hand as with a mashie. Do not get the idea that all this means dispensing with friends. Friendship is not conversation. The things the deaf can do to reclaim the waste places of life, and find happiness in doing, have another rare quality. They are a substitute for conversation in a quite different way. They enable him to account for himself to others, to acquire a new interest in the eyes of his friends, to win a consideration that his amateur performance as a listener will not give. Thus I find myself at fifty-four busy and happy, with a very satisfactory "expectancy" allowed by my insurance company, with a life packed full of the most exciting and enthralling things to do, and wondering whether I am going to have time—even if I realize that expectancy—to do them all.

### ORGANIZED SOCIAL SERVICE FOR THE DEAFENED.

It is obvious that any sustained effort to benefit deafened people along social service lines must be through proper organization. Formerly no institution work was attempted for the social and economic betterment of the deafened, except through institutions maintained for deafmutism. It so happens that the psychology of the deafened person with speech differs so widely from mutism, or deafness without speech, that it becomes well nigh impossible to carry on any social service or educational work which contemplates a combination of the two conditions. This fact has been fully proven and needs no further discussion. The balance of this paper will have to do chiefly with social service work for deafened people or those with speech.

During the last ten years organizations have come into existence with the purpose of solving these problems. These

organizations have assumed various names, like leagues for the hard of hearing, speech reading clubs, etc. Community work for the deafened would seem to be a suitable general term under which the various subdivisions of social service work may be outlined. The ideal organization, in my judgment, is one that would be based largely upon the club idea. First, a permanent home, suitable for carrying out the necessary activities. Second, with trained social workers in charge. Third, independence and self-maintenance on the part of the members, so far as the actual club expenses are concerned. Outside help should be secured covering the expenses of education, vocational training, employment, recreation, charities, research and preventive treatment. Unfortunately, during the earlier stages nearly all organizations have been obliged to receive donations in order to successfully become established. Let us briefly define an ideal community center for the deaf. Ideally, such a center should undertake to do for deafened people service similar to that which is so successfully done for the blind. In New York City the community center for the blind centers around an institution known as "The Lighthouse for the Blind," which is a large home or clubhouse, which contains rooms for the various departmental activities. This would be an ideal arrangement for a community center for the deafened, which should primarily be a clubhouse where deafened people could carry on the various activities. Such a building might include rental quarters for private teaching of lip reading. In some cities in America the Municipal Community Chests have provided the headquarters and equipment. In the formation of a local unit three types of individuals should, if possible, unite—the otologist, the deafened educator and the deafened social workers, in a combined effort to rouse the deafened man and woman out of despair and make them over again into alert, happy, hopeful and self-supporting citizens. Just get a group of the deafened together and organize. The American Federation of Organizations for the Hard of Hearing is prepared to furnish the necessary organization literature.

EDUCATIONAL (LIP READING TRAINING).

Inasmuch as Mrs. Nitchie, in her paper, will cover the entire field of lip reading education, we will be content in merely

urging this procedure and suggesting that otologists make it a routine matter of advice and counsel to their patients. Even though hearing devices are employed, lip reading should be developed in every case, so far as the individual is able to avail himself of this valuable art. It is particularly urged that in the early stages of deafness patients commence the study of lip reading in order to become proficient before the time has arrived when it becomes actually necessary.

EMPLOYMENT BUREAU.

One large department of such a service should be the employment bureau, which should be in charge of trained vocational counselors and placement workers, capable of advising applicants for positions in accordance with the type, degree and progressiveness of his deafness, his physical condition, his education and his temperament. The employment workers in the New York League for the Hard of Hearing make a careful study of each individual case, making use of a card index system which becomes permanently available, and which covers every phase of the applicant's condition. The following are among the questions asked:

1. The age of the individual at the time he is forced to consider rehabilitation.
2. The degree and the type of deafness at the time he recognizes the need for rehabilitation.
3. The finances of the individual and his dependency.
4. The willingness of the individual to accept the aids, such as lip reading, hearing devices, etc., that are available.

The placement workers are obliged to overcome, first, the inadequate facilities for training in a wide range of occupations; and second, the unapproachability of employers. The rehabilitation of the deafened junior is a comparatively simple problem. He is trained in lip reading, equipped with a hearing device, if necessary, and then trained for a vocation where any increase in his deafness will not be a handicap. These occupations include most forms of machine operating, all branches of the sewing trade, all of the agricultural pursuits, some of the professions, and many kinds of business. The exceptions include telephone, teaching (except lip reading), telegraphy, stenographic work, salesmanship and cashiering.

The league has advised training to some of its younger applicants in printing, show card writing, embroidery machine operating, designing and office machine operating, such as the billing machine and multigraph.

The rehabilitation of the person who has engaged in some definite occupation for many years is a more serious problem. Thirty-seven per cent of all the persons who apply to the bureau are forced to change their vocation. For them, advice regarding the utilization of part of their former training and experience must be given, suggestions for proper training and the place to take it, and information regarding those occupations which might be harmful. To those with subnormal hearing any remnant of hearing is most precious; hence, occupations surrounded by unhealthy conditions, such as are found in refrigerating plants, subways and tunnel construction, must be avoided. Positions requiring standing must be avoided by deafened persons who are subject to vertigo. Let us cite the histories of a few cases that have been handled by the bureau during the past year:

1. A nurse with slight deafness. Sought advice from nurses' agencies, doctors and friends. Sought advice at the League. Trained in lip reading, told to give up nursing, given a job where she would not be too fatigued to study lip reading at night; arrangements made for training through the Bureau of Rehabilitation of the State Department of Education, in X-ray operating. Secured a position in a hospital. She wrote: "I was prepared to have a hard time of it, but now scarcely feel that my job is work. I have found it good to combine X-ray with laboratory work and have taken a course in bacteriology. It is not only working, but also the planning to do more that makes me feel a bit more useful again. I am glad to tell you that I can do the work and can also enjoy it. Please accept $5.00 for the League from my first salary.—N. G."

2. Young man, 22. Total deafness after mastoid operation. Clerk in insurance firm before. Advised him to learn all about office machinery. Finally took advice and secured employment in three weeks' time.

3. Man, 50. Very hard of hearing. Uses a hearing device. Yacht captain for seventeen years with the same employer. Lost job not because of deafness, but because man had given

up yacht. We have advised this man, who has some savings, and a wife with perfect hearing, to open up a small shop in a suburb, such as a hardware store, where the counter work could be done by his wife and the handyman work and mechanic work be done by him. In order to convince him that people of normal hearing would accept him on the strength of his ability in spite of his deafness, he was placed in a job as a mechanic for a time.

In many instances a new occupation has to be learned, and the applicant must be given a course in vocational training. In such cases the placement workers, having made a complete study of the individual, are able to advise the kind of occupation for which he is fitted. He is then given such schooling as may be necessary to prepare him for his new job.

An important element in the employment for the deafened is the ability of the placement workers to convince employers of the availability of this type of employee. In the New York League this has been so successfully done that various employers have shown their appreciation by calling for recruits from deafened sources. He is told that the deafened man concentrates better than the hearing; that he is more efficient at general routine work; that he accomplishes more work in less time, for the reason that he does not engage in conversation going on about him. Furthermore, he is more appreciative of his job because he recognizes his limitations and does not seek constant change. In short, his redeeming features, to be mentioned to his employer, are as follows:

1. Highly developed power of concentration.
2. Keen observer.
3. Quick mental movement.
4. Does not waste time in chatter.
5. Remains in position longer than hearing individual.
6. Master of detail.

Of late the demand of employers has been so great that our placement officers are carrying a newspaper advertisement urging deafened people to apply for positions.

VOCATIONAL TRAINING.

Vocational training whereby deafened people may be taught new occupations should be urged in all cases where the victim can no longer follow his usual vocation. In the larger cities

there are special organizations for vocational training along all lines of endeavor. In New York City the Bureau of Rehabilitation of the State Department of Education pays for the vocational training of our deafened individuals in the existing schools of the city. Thirty-four States in the Union offer rehabilitation facilities to the physically handicapped. It is up to the organizations for the deafened and the otologists to make use of them for their cases. In smaller cities the organizations should undertake to find training opportunities for their people. If possible they should provide scholarships in trade schools that would cover traveling expenses and board if necessary. Above all, avail themselves of the city, State and Federal facilities offered and demand them where there are none. Whatever else he may do in preparing for a new job, he should certainly persist in his lip reading studies.

HEARING DEVICES.

Dr. Walker is to discuss the question of mechanical aids to hearing; hence I shall confine my remarks upon his subject to a few brief comments regarding certain important information recently gained. In general, it may be stated that great progress has been made during recent years in the development of hearing devices, particularly in their special adaptation to varying and different uses. For instance, until recently, the executive secretary of the New York League for the Hard of Hearing, Miss Annetta W. Peck, has been entirely unable to make use of telephone conversation. About one year ago she found that by using the phonophor applied to the telephone receiver she was able to hear telephone conversation perfectly. The writer converses with her frequently and has been amazed at the power of the little instrument. The Phonophor Company produces four or five varieties of phonophor, but the one most effective for telephone service happens to be the cheapest one of the lot. Stethoscopes with large mouthpieces (accumulators) can be purchased at Knauthe Brothers, New York City. These might be of service to hard of hearing doctors in examinations of the heart and lungs.

Mr. Storey's comment, previously referred to, calling attention to the fact that hearing people frequently approach the deafened when they see them using the hearing device, is worth repetition at this time. The various manufacturers of

hearing appliances are more than willing to undertake any problem which tends to improve their quality and effectiveness. A recent experience has brought to light a new phase in the hearing device problem which promises to bring untold happiness to many deafened individuals. This knowledge may be in the possession of many in this audience, but the writer's knowledge is of very recent origin. Almost by accident some deafened people have been induced to test the radio with wonderful results. In one instance a deafened man was urged to try it, but for days and weeks refused to even attempt to make use of the radio, only to be enraptured by his first day's experience with this marvelous invention. His letter written at the request of the writer will best tell the tale.

"Dear Dr. Phillips—Knowing of your great interest in the cause of humanity, and especially in those who are hard of hearing, I desire to tell you of my wonderful results from the use of the radio.

"I have been deaf for 45 years—have been deprived from intercourse with my fellow men—deprived from the pleasure of attending lectures, concerts, church. Now, however, that, thank God, is ended, and also ended for all those who are similarly afflicted.

"My outfit is not a very wonderful one—made by the Radio Instrument Company of Washington, D. C., and makes use of six vacuum tubes, yet by 'plugging' in the first stage of amplification I hear perfectly.

"I had had my radio but a few days when on Easter Sunday I listened in to the service conducted at the West End Presbyterian Church, heard distinctly, plainly, clearly, the opening organ voluntary, solo and quartet singing, reading of the lessons, prayers, a wonderful sermon, installation of new members and the benediction, and please believe, it was, to me, a God sent benediction. I say it manfully and without shame or fear of ridicule, the tears of joy streamed down my face. Last Sunday I heard the entire service at St. Thomas' Church —in the afternoon the services conducted by the Bedford (Brooklyn) branch of the Y. M. C. A., by the Rev. Dr. Cadman, who stated there were nearly 1,000 men in attendance, and when this congregation sang that glorious, inspiring hymn 'Onward, Christian Soldier,' the music being played by the

Glory Trumpeters, you can imagine the effect it all had on a poor soul who has spent forty odd years in the 'silent land.' In the evening I listened to a concert by the Capital Theater Orchestra, and the ending of a perfect day was a fine organ concert. I want to add that one number of the theater concert was the 'Melodie' in F, by Rubinstein, played as a 'cello solo. I've heard, off and on, parts of this composition all my life, but never before the entire piece, until it all came to me over the wire, the deep bass notes of the instrument sounding like a beautiful pipe organ.

"I have, so to speak, by means of the radio, found a new heaven and a new earth and am just beginning to lead a normal life. God grant me many years of life in order that I may enjoy, to the utmost, my new found joy and happiness.

"If all this wonderful salvation has come to me it can also come to others in affliction, and I shall appreciate it highly if you will tell me how best to go about spreading the gospel of Saint Radio, as I cannot keep it to myself, but want to shout it all from the housetops for the enlightenment of the world.

"Please feel at liberty to use this epistle in any way desired, so long as it will do some slight good for those who may be afflicted and whose souls are crying for help. Your friend,

"WARREN POND."

Mr. Warren Pond is at the head of the well known firm, William A. Pond & Company, music publishers, of New York, and is evidently a case of otosclerosis.

Since receiving this letter several people with varying types of deafness have been requested to make tests of the radio in an attempt to determine just what types of deafness may be reasonably expected to make use of the radio. The following letters relate the experience of other people:

"Dear Dr. Phillips—You asked me to give you some account of my experience in listening to Mr. Pond's radio. I have tried it twice, once on April 24, and again April 29, when my father went, on Mrs. Pond's invitation. His experience is also interesting; so you have mine, otosclerosis of 38 years' standing, and my father, C. C. O. M. of 35 years' standing. He is quite hard of hearing now, while I have practically no hearing at all, unless hitched to the most powerful devices made.

It is impossible in my own experience to separate the emotional and imaginative from the scientific; but I think you are as much interested in the one as in the other. When, about ten years ago, I dismissed my last pupils in piano and harmony, I slammed the door on my old life and plunged into earning my own living in an entirely new field. So completely have I forgotten that I was ever a musician, and identified myself with social work for the deafened, that I have wondered whether there really was sufficient magic in things heard to justify people in spending hours and hours of precious time sitting around a concert room or an opera house. You can judge therefore of the emotional effect of hearing over the radio the tremendous and thrilling voice of my own beloved piano, not painfully incomplete, but full, rich and satisfying as in the early days of my deafness before I ever dreamed that music would have to be left behind and its glories translated into some other form of human activity and service.

"The repertoire of the piano does not change much, and from old experience I could hear all variations of touch and even the errors of the young player's performance. I heard all announcements surprisingly well, getting names better than titles, and later, in the baseball reports, the names of clubs better than those of individual players.

"On the second occasion I heard, besides announcements and short speeches, songs by sopranos, a tenor and a baritone, orchestra and organ. The latter was particularly successful.

"On both occasions I found my head noises slightly increased —a sort of escaping steam sound—and that my head ached slightly for a time, but my speaking voice sounded clearer to me and my hearing definitely better. Of course, hearing in my case does not mean the naked ear, but hearing familiar voices at their customary pitch and volume with the same hearing device as usual. After two days this continues.

"My father, on the other hand, heard almost too much, just as Miss Samuelson did, and as he does not like the electric hearing devices much at the best of times, he was making all sorts of little experiments in bone conduction, which is more comfortable to his thinking. But the day following he was much deafer, so that we had to raise our voices very much more than usual—in fact, it was all we could do to make him

hear. This was better the next day. However, he is going to be a fan, I can see. Hoping that this will be of some use to you, I am, sincerely yours,

"ANNETTA W. PECK."

"My dear Dr. Phillips—Here's a statement of my experience with Mr. Pond's radio. We listened in on piano selections, several soprano solos and a male voice announcing commission market quotations and baseball scores. I found the speaking voice by far more agreeable. When the radio was tuned at a pitch comfortable for Miss Peck—that is, so that she could hear the piano as an actual piano—it was a most violent noise and tin canny. The vocal selections were ever so much better. During one selection there was a crossed wire, and I could clearly hear the vocal selection with a jazz band and coon shouter. It seemed as if I was hearing one in one ear and the other in the other. When the radio was tuned so that it was comfortable for Mr. Pond it was agreeable to my ears. (I guess that you will agree that Mr. Pond is a bit more deafened than I.) The voices were all clear and distinct.

"The day following I was walking through the tunnel outside of Gimbel's. I heard a phonograph just as clearly as if I stood alongside of it. I investigated. In the store near one of the entrances I found a radio with the horn attachment. I should say that I heard the music (as much, which is rarely true of a naked phonograph) at a distance of at least 150 feet through walls and windows. I don't hear our own victrola at home unless I am in the room in which it is playing.

"The possibilities of radio as a reeducator of hearing seems great to me, but raises one great question in my mind. I really am a fan and wouldn't want to take the joy out of the game for the world. Very sincerely yours,

"ESTELLE E. SAMUELSON."

Miss Annetta W. Peck I know to be a case of otosclerosis and her father to be one of chronic catarrhal deafness. These experiences certainly serve to determine the fact that the radio has brought marvelous possibilities of experience for our deafened friends. It promises to become a source of educational value, even in the schools for deafmutes. So enthusiastic have a few members of the New York League become that the whole place is being installed with radio equipment for the

benefit of its members. Several have already remarked that they propose to attend church every Sunday morning and evening at the League headquarters and to listen to lectures and concerts at other times.

Some of our workers have found much benefit from an appliance called the Auricles, manufactured by the William V. Willis Company of Philadelphia. This instrument is quite similar to the English made instrument known as the Aurolese Phone, but possesses the added advantage that it can be purchased for about $10.

RECREATION AND AMUSEMENTS.

It is surprising how much real pleasure may come from organized efforts to promote certain types of amusement and appropriate recreation measures for deafened people, particularly in groups. Ordinarily, the deafened person gets his chief amusement from books, moving pictures or from some fad. It is possible, however, under trained leadership to develop a variety of amusements for men and women of mature years, such as card parties at headquarters, for which clubhouses should provide opportunities. Younger people become very much interested in theatricals of the pantomime type, and when possible they are exceedingly fond of dancing. Recreation in the form of club hikes, with lunches, botanical, nature and other available studies.

PREVENTIVE DEAFNESS.

Within the domain of social work, under otologic supervision, can well be placed opportunities for studies along the line of preventive deafness. Naturally these studies will have to do largely with child life. Dispensary and hospital treatment for the deafened has up to date failed to conduct specific investigations along the lines of preventive deafness in children. It is true that certain well known causes are carefully attended to in the large clinics. By this I refer to the removal of tonsils and adenoids and similar measures, but to give a specific study to children who already show signs of deafness and to treat these children with the sole purpose to prevent further inroads upon the hearing function has never been generally adopted. Dr. James Kerr Love of Glasgow*

*Volta Review, November, 1921.

has done some pioneer work along this line, and Dr. Franklin W. Bock has attempted the same measures in the Rochester public schools.

Since October, 1922, it has been attempted under the auspices of the New York League for the Hard of Hearing. The plan has been about as follows: No children have been subjected to examination and treatment except those who have been secured under League auspices and by our professional workers. In every instance full records are obtained at the League covering the name, age, home conditions and all other possible information is obtained and recorded. This information is not only recorded but filed and a duplicate record card is sent to the doctor. The following is a sample of the card:

CLINIC FOR THE PREVENTION OF DEAFNESS IN CHILDREN

NEW YORK CITY

Date.....

Name .....
Address .....
Age .....
Degree of Deafness .....
Cause of Deafness.....
Speech .....
Illnesses .....
Deafness, first notice.....
Head Noises .....
Earaches .....
Tonsils and Adenoids.....
Where treated .....
Deafness in family.....
Parents' name.....
School .....
Grade .....
Teacher .....
Remarks .....

Registered by:

The New York League for the Hard of Hearing, Inc.
126 East 59th Street

The patient is then given a complete otologic examination by competent otologists. This examination is supplemented by such other forms of examination as the symptoms may suggest. In some instances even a Wassermann test has been made. The child is then placed under treatment—nasal and pharyngeal abnormalities and diseases eradicated, advice and counsel is given as to diet and home life. The prevention of colds and proper aural guidance during and after colds. So far no efforts have been made to enter the field of pathology. Furthermore, the number examined up to date is too small to enable us to form definite and permanent conclusions. In all, about 60 children have been under observation and have been given a total of about 500 examinations and treatments. A preliminary report will probably be published in the near future. Suffice it to say the results of these examinations and treatments have more than warranted the efforts which have been put forth, and it would seem that the time has come for the opening up of departments for research in the prevention of deafness in our ear hospitals and clinics. Furthermore, it should become one of the leading features of social service work in the field of otology. Particularly, in our very large cities for the treatment of ear diseases, there should be a distinctive department devoted entirely to the subject of preventive deafness. I venture to report a recent case coming under my observation which serves clearly to show the need of a more careful study of hearing conditions in children, with a view to the prevention of deafness, and second, to the proper treatment of children showing symptoms of defective hearing. A girl ten years of age showed symptoms of gradual loss of hearing for about two years. The mother had taken her to one or two general practitioners, who failed to discover any cause for the affection. In school she had commenced to fall back in her studies, and the teacher had finally advised the mother to place the child in the public school for the deaf. It is evident that no competent otologist connected with the school system had ever examined this child. It was found that her hearing was so defective that it would be impossible for her to continue her studies by regular school methods. Upon examining the canals they were both found to be tightly packed with cerumen, and after its removal she heard as well

as ever. This case may be a rare exception, but it serves to emphasize the laxness in our present methods of examining school children and emphasizes the fact that there is a wide field for investigation along the lines of prevention in the domain of aural diseases.

This I have briefly outlined various phases of social service work which may well be attempted by individual organizations. After two or three years of rather intensive study of the problem as undertaken by the few organizations in the larger cities, I became convinced that this type of enterprise should be encouraged by the formation of a national organization, for the following reasons: First, to provide a central body which would have the control and management of an annual convention in the interests of local organizations. Second, for interchange of views and for development of methods, and to encourage the formation of local organizations throughout the country. This organization is now on a well founded basis, under the title of "The American Federation of Organizations for the Hard of Hearing." The fourth annual convention was held in the city of Chicago, June 18, 19 and 20. The third annual convention was held in Toledo in June, 1922, and over 250 delegates registered from various parts of the Union, all the way from Boston to San Francisco. At the present time fifteen constituent bodies are in affiliation with the national organization, and many more are making ready to join or are in the process of formation. There are two foreign local associations—one in Glasgow, Scotland, and one in the city of Prague. The Volta Review has become the official organ of the Federation, which has its headquarters in the Volta Review building in Washington, D. C.

In conclusion, I would say that social service work for the deafened offers a wide field of actual service to humanity on the part of the otologist. Every large town or city should have an organization to carry on this work. The otologist, recognizing the timidity of the deafened in public matters, should lead in the formation of these local units. Many of the local organizations throughout the country have come about in just this manner. Upon entering this field the otologist opens the door of his life to the very highest realm of actual service to humanity. It calls for unselfish expenditure of time

with little to be gained along the line of financial emoluments. For in the main he must give of his time freely. It is a disinterested service and brings little fame. It is the fulfillment of the provisions of the Hippocratic oath in its very highest sense.

NOTE.—Comments herein made from papers of Mr. Starling W. Childs, Mrs. George L. McAlpin, Mrs. Julia E. Johnsen, Mrs. Robert C. Morris, Mr. John de Raismes Storey and Mr. Earnest Elmo Calkins are practically in the exact language of the individual authors.

LV.

## AIDS TO HEARING.

By Gordon Berry, M. D.,

Worcester, Mass.

On the 4th of August, 1922, thirteen million telephones were silenced while the nation mourned one of her greatest. Poor and potentate the world around sent messages of sympathy and condolence. As the sun was setting on Beinn Bhreagh, the highest spot in Eastern Nova Scotia, there in the solid rock the body was laid to rest while the stillness was broken by the singing of Stevenson's "Requiem":

> Under the wide and starry sky,
> Dig the grave and let me lie;
> Glad did I live and gladly die,
> And I laid me down with a will.

Five months have passed, and on the 3rd of this month (January, 1923) his widow, alike the cause and inspiration of his wondrous accomplishments, went to join him. Increasingly as the years swing around will man realize his debt to Alexander Graham Bell and Mabel Hubbard Bell. To some who are intimate with his manifold activities it seems that not in his remarkable geographic research, not in his exhaustive statistics on heredity, not in his pioneer work with aeroplanes, not even as the inventor of the telephone, but in his constructive championing of the deaf, lies his first claim to the gratitude of posterity. The proximity of their deaths, and their intimate association with all progressive effort for the deaf make it fitting that we refer to these two and their early struggles. We can thus render homage, and at the same time through their lives review the history of work for the deaf during the past fifty years.

Dr. Bell's grandfather gained fame in Scotland as a teacher of elocution and speech training. His father, Mellville Bell, invented the famous "universal alphabet" now used in teaching phonetics. It was Dr. Bell's boyhood enthusiasm which cre-

ated a rubber skull that could say "mama" and that so trained a pet dog that when Bell's fingers pressed successively on different parts of its larynx, the dog could be heard to say "How are you, grandma." When (at the age of twenty-four) Bell first came to Boston in 1871, Miss Sarah Fuller was at the head of the Horace Mann School. She early asked him to teach her workers. Then he spent three months at the Clarke School for the Deaf and seven weeks at the Hartford School. He rapidly became an authority in speech training. To his Boston school came young Mabel Hubbard, whom he later married.

A few years previously (in 1864), at four and a half years of age, this little girl had become totally deaf from scarlet fever. Her father learned that the Germans were successfully teaching their deaf by the oral method, but that in this country only the sign language was used. Mabel seemed destined to lose her speech as well. Here in Rhode Island Governor Henry Lippitt had a deaf daughter, Jennie, whom the mother had been teaching by the oral method for six or seven years. With Mrs. Lippitt's encouragement and advice, Mrs. Hubbard now began to teach Mabel, and so successfully that at six years of age she and her friend Jennie Lippitt were brought before the legislators on Beacon Hill, where the question of the founding of an oral school for the deaf in Massachusetts was hanging in the balance. The Solons could not but yield to the convincing proof these two little girls gave, and in 1867 a charter was granted the Clarke School for the Deaf at Northampton, the pioneer of the oral schools in this country; for the Hartford School, founded in 1817, and other more recent institutions for the deaf were all using the sign language method which Gallaudet had brought over from the Abbe Sicard's school in Paris. In the meantime Isaac Rosenfeld had been seeking aid for his deaf child. The final result of his efforts was the opening of the "New York Institution for the Improved Instruction of Deaf Mutes" in 1867, and nine years later the "Rhode Island Institute for the Deaf" was founded here in Providence. To Gardner Green Hubbard, Mrs. Mary A. Lippitt and Isaac Rosenfeld belong the credit for sponsoring these splendid institutions, but not more than to Harriet B. Rogers in Northampton, Sarah Fuller and Mary H. True

in Boston, J. W. Homer in Providence and Bernard Engleman in New York, the pioneer teachers who built the foundations of our structure.

The second chapter comes with Dr. Bell's efforts to help these deaf children. He had found by tests that many had some hearing. By utilizing this little, he was much helped in getting them to place their sounds correctly. He used speaking tubes with profit and was anxious to discover some electrical means of magnifying sound that he might still better reach them. With Dr. Clarence J. Blake's help he fastened a stylet to the ossicles on a model of a human ear and was able to obtain a record on a rotating drum of the membranous oscillations made by the spoken voice. He called this the phonautograph, and had the deaf children use it. He found that a small resonator box containing tense violin strings when worn next to the child's chest would give tactile warning when a bell or horn of an approaching vehicle on the road spelled danger. Then he became convinced that some resonator might be so adjusted as to cause an undulating current in an electric wire, and that the reversing of the process would pick it up in the receiver end as duplicated sound waves. This was his initial conception of the telephone. At about this time (1874) Bell was working as a side issue with his "harmonic telegraph," in an attempt to send six or eight Morse messages on a single wire at the same time without interference. The mechanical work was being done by Thomas A. Watson. With characteristic perseverance, Bell worked through that winter and the following spring. He was using springs of different tension, something like tuning forks, and trying to have the electrical wire transmit a selective impulse as sound to similar springs in an adjoining room. Watson describes that momentous afternoon of June 2, 1875. "We were hard at work at the same old job. One of the transmitter springs I was attending to stopped vibrating and I plucked it to start it again. It didn't start and I kept plucking it, when suddenly I heard a shout from Bell in the next room, and then out he came with a rush, demanding 'What did you do then? Don't change anything. Let me see!' The make-and-break points of the spring had become welded together, creating just the necessary electric circuit for transmitting sound." Fortunately

it was the genius Bell who was in the other room and who heard the faint ticking on his end of the wire. There was born his telephone. A similar good fortune in 1900 destined that Lee DeForest should be working near a Welsbach burner when testing electrolytic detectors for wireless signaling. He noticed a brightening and dimming of the gas light as the spark transmitter was operated. From this came the audion amplifier, which permits telephonic conversation across the continent, enables a vessel to come into New York Harbor in a fog, and promises to play an important part in electric hearing devices of the future.

It was not many days before Watson's fevered activity under Bell's direction was able to make the crude transmitter through which the first telephone message was delivered—"Mr. Watson, please come here, I want you." Then came endless experiment and adjustment. Finally they were ready to make an outside test. Permission had been gained from the Walworth Manufacturing Company to connect up with their privatet telegraph wire two miles long, running from Boston to Cambridge, and on October 9, 1876, Watson first heard Bell's historic call "ahoy, ahoy," over the long distance telephone. Perfecting the telephone consumed so much of his time that Dr. Bell had to give up other activities for the time being. But his interest in the cause of the deaf was just as active, as witness his letter to his mother in this same year (1875) when convinced of the success of his invention: "Now we shall have money enough to teach speech to little deaf children." And it was Mr. Hubbard and Mr. Thomas Sanders, fathers of two of his deaf pupils, who furnished the financial backing to start the telephone. Thus inseparably is the telephone connected with deafness. And later this same telephone was to be the basis for the electric hearing devices which we are now using.

With the establishing of the Bell Telephone Company and the clearing up of legal difficulties following infringements of his patents, Dr. Bell was free to enter such fields of endeavor as his desire directed. In 1878 he moved to Washington. Here he was always ready with voice and pen to enter the lists vigorously in behalf of any enterprise that tended to

ameliorate the isolation of the deaf or better their training. Here he conducted his extensive researches in hereditary deafness. Of peculiar interest was his study of the colony at Gay Head on Martha's Vineyard. The excessive deafness among the inhabitants there, together with accurate historical records of their consanguineous marriages, led to his important conclusions against the marriage of the congenitally deaf. He called together the first convention of oral teachers for the deaf, which met in Worcester, Mass., in January, 1874. This group kept itself intact under his leadership and was stimulated by his summer gatherings at Lake George until it won recognition at the twelfth convention of the American Instructors for the Deaf (sign language group) and was incorporated in 1890 as "The American Association to Promote the Teaching of Speech to the Deaf." Dr. Bell was its first president and endowed it with a personal gift of $25,000. When the French Government awarded him the Volta Prize of $10,000, because of his discovery of the telephone, he founded the Volta Bureau and gave it this money for the equipment of the Volta Laboratory. Here were invented the graphophone, wax disc records and an improved phonograph. This prize gift and his subsequent donations made a total of about a quarter of a million dollars. In 1909, at the founder's suggestion, the Volta Bureau with its endowment was presented to the "American Association to Promote Speech to the Deaf." Today this Bureau publishes the Volta Review, has a library which is the most extensive repository of literature concerning phonation and the deaf in existence, serves as a rallying point for new enterprises for the deaf and as a patron of all who will conduct research in their behalf—in a word, is the central agency for the deaf in this country.

With this cursory review of the early work for the deaf, let us turn to the present day aids to hearing that Dr. Bell's efforts and those of others have contributed. The subject naturally divides itself into a consideration, first, of the mechanical devices to aid hearing that are on the market; second, lip reading as a very important help; third, of the cooperative work now being done in this country for the deaf.

There are many mechanical contrivances purporting to make the deaf hear. They all have something in their favor.

First, there are the various forms to be placed in the auditory canal to fill in a perforated drum membrane. Considerable skill is attained in applying the piece of rubber or membrane. By actual test the hearing may be better after its insertion. Such appliances are to be condemned because they are not intelligently used, and because the unsuspecting victim almost invariably injures the canal or aggravates or creates an active suppurative process. The second type evidences itself in some form of modification of the hand cupped around the ear to facilitate hearing. The Globe Phone Company advertises nine forms in its circular, all of which are on the basis of a horn or tube which will catch a larger volume of sound than the unaided ear and will convey this directly through a confined air channel to the external auditory meatus. This type has the advantage of transmitting sound, exactly as it is received without accentuating certain types of sound and lessening others, and is found particularly serviceable in the aged deaf. In this group may be included quite a different mechanism, which relies on bone conduction for hearing. The best is a hard rubber fan held against the teeth. Sound impressions are caught on it and carried through the teeth to the skull and so to the internal ear. The third type is being steadily perfected and, by reason of its finer control and better adaptation, is gaining supremacy. I refer to electric hearing devices.

Priority in manufacture of the first ear phone appears to be in doubt. I am informed that C. W. Harper first brought out his Oriphone in Boston in 1902, and that Miller Reese Hutchinson of Mobile, Ala., first produced the Akouphone in the same year, changing the name later to Acousticon. The Globe people were also pioneers. These instruments have been made in many sizes. The smallest of which the writer has heard had a receiver which could be inserted almost out of sight in the ear canal, a battery one inch long and three-quarters of an inch wide, and a transmitter the size of ten cent piece which could be worn as a lapel button. Such small sizes are impracticable. An enumeration of the different standard ear phones on the market in this country will give a concrete idea of the growth of this industry. They are the Acousticon, the Gem, the Globe, the Harper, the Magniphone, the Mears, the Midget Phonophor, the Opera Type Miss Lucy M. Bruhn

brought last fall from Germany, the Portophone, the Potter, and the Williams Articulator.

The mechanism of the telephone and of these instruments depends upon what might be called a loose connection which has been gained by the insertion of many carbon granules or balls into a continuous electric circuit. These are enclosed in a chamber or in many little cups, and the movement of the diaphragm against which they rest makes a larger or smaller number of electric contacts among these balls. This varying resistance gives an electric fluctuation: Bell's "undulating" current. Occasionally a carbon ball gets caught, making a permanent contact, with a consequent hissing sound. This and other imperfections manufacturers are trying to correct. The battery for furnishing the electric current has been improved and simplified; unnecessary parts have been eliminated; the electric cord has been perfected. One worker (Charles W. Harper) spent two years in determining the best material for the diaphragm. The war has contributed its quota. Earl C. Hanson, by experimenting with the audion amplifier in his wireless work, was able to help men at listening posts hear the enemy's plans, use a tree as a wireless receiver, enable a flier to get messages from the ground. His deaf mother had made him anxious to make some contrivance to help her, as those already existing did not seem to serve. With the cooperation of the Western Electric Company, which controls the audion patent, he made the Vactuphone for the Globe Company and thus introduced a new principle into the science of ear phone manufacture.

An ear phone transmitter lends itself to considerable adjustment. Two factors are present. The writer is told that the size and texture of the diaphragm determine the amplitude of its wave length, while the stiffness of the diaphragm and the controls at circumference and center determine the amount of its in and out swing. The first factor regulates the pitch which is transmitted through the cord to the receiver and the ear; the second determines the intensity of that sound. As we know experimentally that certain patients, though deaf, can hear far more clearly at certain pitches than at others; and on the other hand that where there is a partial nerve deafness superimposed a greater intensity both tires the ear and

drowns out the sound; it is clear that these two factors of distinctness or pitch and volume or intensity must be intelligently controlled. Does not this suggest where the otologist may contribute by determining the degree and type of deafness? In the research laboratory of the Western Electric Company one can so carefully test an ear with their graded sound producing appliances (such as the audiometer) as to chart the acuity of hearing for differing pitch and volume.

How much gain can a deaf patient expect from a properly adjusted ear phone? A conservative estimate would triple the range of hearing in the average catarrhal case. This is a real gain. Manufacturers and deaf alike say that one must train himself to use the instrument. Certain types of noises are much magnified, others are scarcely audible. The user learns how to adjust the rheostat, dimming the loud noises and increasing the soft. Adventitious noises which the instrument accentuates he learns how to pay no attention to. In nerve deafness these adventitious noises trouble more, and here the ear phone offers no aid but jangles and tires the ear. Where there is a catarrhal as well as a nerve deafness, an instrument which accentuates detail without undue volume will help the catarrhal phase and not tire the nerve phase. It is claimed that these instruments improve hearing. There is logic in this contention. We know that disuse of a nerve causes its atrophy.

What is our custom when we are persuaded that we cannot further help a deaf patient? Do we ask him to see whether lip reading will help and bid him go to the store and secure an ear phone for one week's trial? Can we not be more accurate and definite in our recommendations? One manufacturer suggests the possibility of grading the instruments into standardized groups. He would make some device whereby the otologist can test the patient in order to discover which ear phone will fit the given case. We could then prescribe an instrument as the oculist prescribes glasses. The ear phone is still in its infancy. When it reaches full stature it is conceivable that such an Elysium awaits the hard of hearing patient.

We have mentioned how lip reading for children received its first impetus in America. Schools for deaf children sprang up in every state, and a tremendous amount of good is being done by them. It is not generally realized that from an institution

like the Clarke School for the Deaf an average of at least one goes out each year to enter high school on equal terms with the hearing children, and that one-half of these go on through college. The handicap is present, but with the use of other faculties than the one impaired sense of hearing, graduation with honors is attained, and a successful life in business or some other pursuit is the result. It is of the adult deaf that the writer would speak. This is a different study. Education has been acquired, the power of speech has been fully developed, contact with the world and one's fellow men has been intimately established. That contact must be preserved when deafness has come. Is the ear phone the only means?

Chance actively identified the writer with the Army Lip Reading School for the returned war deafened soldiers during the most of its fifteen months' existence, first at Cape May, N. J., then at Fox Hills, Staten Island. In his candidate's thesis before this same society a year ago, he made this experience the subject of an intimate study. He desires to refer to this work and his conclusions, that we may contrast the benefit to be gained by mechanical means over against the gain from lip reading.

What type of pupil were we dealing with? There were 108 altogether. All but nine were American born. They were young, averaging 26 years of age, and had been of sound body and mind when entering military service. They came chiefly from the so-called laboring class: farmers, laborers, clerks, machinists; and their previous education was low, as but one-fifth had gone beyond a grammar school education, and one-quarter of them were illiterate. The arbitrary qualification for admission to the school was the inability to hear ordinary conversation at five feet. Of the resulting 108 pupils, 32 showed "total deafness," 13 could hear only a "shout," 24 heard more than a "shout" but less than "conversation" at one foot. The extreme types of deafness were auditory nerve forms, while the less severe cases were of catarrhal or suppurative origin. Fifty-two became suddenly deaf, 56 were progressively deafened.

What were the methods employed? Nine of our teachers devoted their entire time to this work. Since these men were

there for the primary purpose of learning lip reading our effort was to give them all they could stand without undue tire. We wanted to saturate them with it. Each pupil had two lessons in the morning of forty-five minutes each and a third in the afternoon of thirty minutes' duration. He had the sole attention of his teacher for the lesson period. She carefully and systematically arranged his work and took him from easy lip sounds to harder tongue and palatal sounds, and then arranged these varying mouth positions and movements in words, phrases, sentences and stories; but the pupil was not conscious of this systematic progression. His sole task was to read his teacher's lips. It was further found best to change the teacher from time to time. This helped him to get used to different speech movements.

What were our early results? Not very good. Failure to see any good to be derived, lack of ambition, fear that learning the art would make them able to support themselves and so relieve the government of having to pension them, despondency, ignorance, laziness, all played their part to create an early inertia which only the persistent zeal, optimism and patience of the teachers could combat. But the inertia had to give way against such pressure.

The big unlettered soldier was being convinced against his will that such practice sentences as "she bought a cap and a map for the baby" must contain profound interest. One man's experience during this stage of transition will illustrate. He started willingly enough, but did not have much stamina and became quickly discouraged. Then he refused to work at class; found every excuse he could to skip attendance. He was referred back to the medical officer for discipline. Neither reasoning nor censure nor the threat of punishment availed. His discharge papers were started and he was taken out of the school. But one of the teachers was not satisfied and gained permission to see what she could do. The bullying, flattery, ragging and entreaty now showered on this luckless man were worthy of a better cause. He sulked, and he yawned; she kept after him. He broke down and cried; she called him a quitter and scolded him. The upshot was that she had that fellow following her around like a trained dog. He worked as though his life depended upon it. His nights were even sleepless from

worry, his eyes became congested from concentrated strain to watch lip movements. A little divine fire had gotten into his soul. He implored his officer to postpone his discharge proceedings, but was told that he had had his chance and now he must take the consequences. So he hurried back to his teacher for more work. The discharge papers came but were pigeonholed until the man had had his full opportunity and the teacher had realized the joy of returning yet another war deafened wreck to useful and happy citizenship. This case is extreme, but it illustrates well many another case of labor and accomplishment and shows how seriously the work was undertaken. The inertia was conquered, enthusiasm followed, optimism prevailed.

All were not hard cases. Many seemed to have an aptitude for lip reading. For most, three weeks of training was sufficient for them to get along fairly well. Some got much delight out of going to dances, where they could see and feel the rhythm of the music and perhaps hear a little. They conversed as entertainingly, the ladies said, as their hearing fellows. When one boy, totally deaf, asked for the day off that he might go gunning for duck in the marshes, we feared that we were taking chances. But he got more birds than anyone else in the party, nor was anyone hurt. Many of the members of our society recall seeing some of the men at the Atlantic City meetings in 1919, and remember how easy it was to talk with them. Some became so proficient that it really seemed they could hear. One man went home on a furlough and it was two days before his people realized his deafness.

We graded them into five classes. Seventy-four per cent finished in the first two gradations, which meant that they could understand over 80 per cent of what their teachers said to them. It is interesting to note that the best lip readers took the shortest time to complete the work. The average time for the entire school was 2.7 months, a remarkable showing when one realizes that this average includes such hard cases as those meningitic, where a definite slowing of cerebration had followed the disease. Our statistics show that the amount of the man's education made practically no difference in his skill as a lip reader, nor did the degree of deafness seem to have a bearing. We were fortunate in being able to follow these

men into civil life through the Federal Board. Reports came back to Washington from all, and it was gratifying to read the enthusiastic reports from the Red Cross and governmental agencies. Some of them wrote that they had to make more than one visit in order to convince themselves that these men did not hear. Many were going into schools of various kinds and a few even into universities. One man hopes to go through Massachusetts Institute of Technology and graduate as an architect; another passed the first year at Worcester Polytechnic Institute and only discontinued because his eyes gave out.

A detailed discussion cannot be entered into here, but I think you will agree with me that the cause of our phenomenal success in the face of such handicaps was not the remarkable work of our specially trained and enthusiastic teachers, and not our military control over the men, but our intensive program. Hard, concentrated effort and continued practice seem essential to the best accomplishment. With such effort our experience shows that all but the mentally deficient can succeed.

This brings us to the third and last phase under discussion: The cooperative work for the deaf now being done in this country through school and guild and association. Tables published last year by the "Annals of the Deaf" show 158 public residential schools for deaf children in the United States, 76 public day schools and 18 denominational and private schools. Canada has 7 schools. In the last number of the "Volta Review" are listed fifty-eight lip reading schools for the adult deaf. The hard of hearing (who for brevity's sake have been classified in this paper as "deaf") have gathered themselves together in associations for mutual betterment and encouragement. There are twenty-four scattered throughout the country. Fifteen of these have become constituent bodies of the "American Federation of Organizations for the Hard of Hearing," of which Dr. Wendell C. Phillips has been president for three years and is now succeeded by Dr. Harold Hays, while Drs. Richardson and Shambaugh have been active in its affairs as directors. Canada boasts two clubs and Scotland one. The New York League has about a thousand members, that in Philadelphia had 803 last June, the Boston Guild 490. Note what the

New York League does where Harold Hays is the leader: Has headquarters occupying one floor of an office building; gives free instruction in lip reading; has a placement and vocational bureau; has industrial, educational and welfare departments, recreation work and nine clubs; and publishes a monthly newspaper. As secretaries or as directors of these guilds, one finds men and women devoting their talents and their lives to the betterment of the deaf in their community. Here the new member, discouraged, perhaps despondent, finds companionship, encouragement, cheer. He learns how and where to secure standard hearing devices, is warned against the charlatan. He joins the lip reading class. He is urged to take active part in the club's affairs. All about him are people who, though deaf, are making their lives count. He realizes he has magnified his loss and that other senses and other attributes can easily make up for this deficiency. Nor is this all. These organizations are becoming an increasing power. Leaders are included in their membership. By local and national organization they are bettering the cause of the deaf throughout our country. Such is the happy fruition of the work begun by Dr. Bell and his colaborers fifty years ago.

Three phases of "aids to hearing" have been discussed. Should emphasis be placed on any one? The conclusion is that the deaf patient should be urgently encouraged to use every helpful agency. He must be reminded that he has lost but one of his senses and that those remaining offer every facility for a life full of joy and service and accomplishment. Through the varied aids at his disposal his lot is becoming increasingly easy. The day is near at hand when he will no longer find himself so handicapped.

19 ELM STREET.

LVI.

# MASTOIDITIS IN THE AGED.*

By Ralph A. Fenton, A. B., M. D.,

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Mastoiditis is relatively a rare ailment in people of advanced age. Now and again cases crop up in the literature, while doubtless many more are unreported, or are included in series catalogued by reason of special complications. Consideration of various hospital and infirmary reports during recent years would indicate between 1.5 and 2 per cent of mastoid cases occurring in persons past seventy. Sprague[1] in a series of 200 reported four over seventy years; one was fatal from erysipelas and septicemia.

The insidious character of mastoiditis in the aged is well shown in reported cases. Classed very frequently before 1890 as septicemia, as brain fever or brain abscess, as pyemia, these cases rarely were considered in the light of discoveries made by von Troltsch, Pagenstecher[2] and others a generation before. Bryant[3] reports a woman of 63, with whom he waited thirteen days after the disappearance of mild unilateral mastoid symptoms, avoiding operation because of apyrexia. Death occurred apparently from streptococcus bronchitis, but on postmortem examination extensive double mastoiditis was found, with an intact tympanic membrane on one side.

Butler[4] cites the case of a man of 84, having acute bronchitis and no previous otitis, admitted in May, 1919, with a temperature of 99, who remained slightly ill for ten days. The bronchitis was due to a mixture of streptococci and staphylococci. On the twelfth day his temperature rose suddenly to 102, without a chill. The left auricle became very tender, and next day there was spontaneous rupture of foul pus into the external auditory meatus. In two days more there was great tenderness over mastoid tip and antrum, and the patient

*Thesis accepted by the American Laryngological, Rhinological and Otological Society.

begged for relief. The mastoid was opened under local anesthesia, with 2 per cent cocain swabbed on and one-fourth per cent injected subperiosteally. Temperature became normal in five days, when a low grade erysipelas supervened, lasting ten days longer. The patient was discharged late in June, 1919, with normal hearing and the wound perfectly healed.

A similar case is reported by Saunders:[5] A retired physician of 70, with no mastoid tenderness, having pain shooting to the vertex and forehead, was advised on the sixteenth day to be operated, but refused until the forty-sixth day. During a two-hour gas ether operation the entire left mastoid was found excavated, with perisinus and epidural abscesses. There was drainage of cerebrospinal fluid for ten days. Although the prognosis was bad, the ear was healed dry in two months. This man remained well save for a slight attack of right otitis until age 76, when his right ear became involved after a nasopharyngeal infection, Shrapnell's membrane being mainly affected. There was little pain, but on operation, which was not delayed, another perisinus abscess was found. This operation took one hour and forty-five minutes under gas-ether. A pure culture of streptococcus capsulatus was found. The dressing was iodoform gauze for six days, then bismuth-iodoform-petrolatum (BIP) paste; he left the hospital in two weeks, and the wound was healed in three. Five weeks postoperative he had a slight vertigo and was unconscious for a short time, but has since been well.

Certain generalizations will be made from brief reports of three cases in our own service, aged 71, 76 and 82; also certain phases of three others, aged 60, 63 and 64.

Case 1.—L. W., widow, 71, seen January 7, 1920, gave a history of throbbing pain in the left side of the head for five days before. On incision of the bulging pale drum there was a free flow of thin yellow pus. Operation next day (temperature remaining 100 to 97, pain unrelieved) under gas-ether, revealed a large pneumatic mastoid filled with necrotic gray granulations. A streptococcus was present in pure culture. The operating time was twelve minutes, from incision to insertion of stitches; the dressing was dichloramin chlorcosane oil, 2 per cent, with a short gauze plug in the lower angle of the wound and two silkworm stitches above. Low grade ery-

sipelas came on two days postoperative, with temperature to 100.4, and lasted six days. This did not prevent the patient from sitting up in bed and lamenting her appearance. She was out of bed the tenth day, and home with the wound healed on the twentieth day. A short strand of silkworm gut, coiled up for use as a drain, was overlooked in one of the later dressings by the interne and remained encysted until its extrusion eight months later from the lower angle of the wound. Hearing two weeks after the operation and thereafter was one-third normal, better than one-fourth normal rating of the unaffected ear. This patient is still alive and well.

Case 2.—J. K., widow, 76, gave the history of an acute right lobar pneumonia ten days before, following closely upon her husband's death. The right ear had been sore for four days; there was intense pain in the side of the head, but little or no mastoid tenderness. Temperature ranged from 100 to 100.6; white count was 11,000. A small amount of thin streptococcic pus came out after free paracentesis. Operation was avoided for one week more, when the mastoid was opened (April 21, 1921) under gas-oxygen, as pain was increasing and drainage seemed inadequate. The entire process, large and pneumatic, was an abscess cavity; the sinus was exposed for a centimeter, and the necrotic process had proceeded to the digastric fossa. Operative time was eleven minutes; oil dressing; two sutures. This patient's postoperative temperature did not go above 99, and she went home in one week. The wound was healed and normal hearing restored in twenty days; the patient is still in good health.

Case 3.—A. L., widow, 82, was totally deaf and complained that her ears had been very painful for a week past. A doctor had advised her to "let them break." She had not slept for two nights; both mastoids were very tender. The temperature was 103.8, white count 16,000, and albumin and acetone were present in considerable amounts in the urine. Double operation was done February 4, 1922, under gas-oxygen. Very hard cortex was encountered on both sides; the cells were large and filled with very foul greenish pus, mixed streptococcus and staphylococcus. On the left side, as much bleeding occurred from a large emissary entering the exposed sinus, tight roll packing was used. Operating time right side was eleven

minutes; left, fourteen minutes; oil dressing; two stitches. Temperature postoperative ranged from 101 to 99; albumin was gone after one week. This aged and infirm woman left the hospital in eighteen days, and both wounds had healed by the twenty-sixth day. Her former chronic catarrhal deafness was of course unchanged. No inflammatory otitis occurred again. This patient died, several months later, from pulmonary hemorrhage during a slight coughing attack in the course of an acute bronchitis.

In the case of B. J. K., man of 60, cardiovascular patient, an acute right otitis had been permitted to rupture the drum membrane behind an almost occlusive exostosis of the canal. For six weeks, until his pain was almost unendurable, he had received palliative treatment in a distant town. At operation, February 25, 1920, the usual large mastoid cavity was found filled with fetid yellow pus. The cortex was very hard, and there was much deep bleeding; operative time, twenty minutes. This huge excavation filled in but slowly, and the ear was not dry for thirty-six days.

The case of L. H., man of 64, is noteworthy because after a delayed operation (1917), at which a very large cavity was found, and after which the previously high temperature came down to normal, the patient was discovered dead in bed on the morning of the fifth day postoperative. Death was due to coronary embolism; the patient was markedly arteriosclerotic.

The case of E. T., widow of 63, is of interest in that it followed by one week a severe tonsillitis, in a person with chronic bilateral posterior ethmoiditis. The very hard cortex masked an extensive zygomatic cell group. Under gas-oxygen, February 16, 1922, her operation took eleven minutes. She was up on the fifth and home on the eighth day, but has had recurrent mucoid discharge from the ear, following upon certain colds. With treatment of the ethmoiditis the aural discharge has finally ceased.

Conclusions based upon so few reported and observed cases must be drawn with caution. Avoidable errors have been checked in our brief series by the collaboration of internists and laboratory experts, so far as possible.

Sudden lowering of bodily resistance, followed by acute bronchitis or by acute exacerbation of chronic bronchitis, may

be noted as a frequent factor in the etiology of mastoiditis in the aged. Preexistent otitis media, perhaps long forgotten by the patient, must not be ignored in the history, when an estimate of possible danger to the mastoid is made during acute otitis of old people. The remarkable discoveries of Wittmaack[6] in mastoid pathology, interpreted in America by Norval Pierce, may explain the easy cure of some elderly persons, while others, after recurrent "colds" and "catarrhal" deafness, suffer at last a ready invasion of the abnormally developed mastoid. Arrest of normal pneumatization and ingrowth of abnormal epithelial masses and vascular channels may have existed in such persons since childhood. Also, as suggested by Jones,[7] these abnormalities and also the flinty cortex and main trabeculæ of advanced age may be simulated by the early aging process which accompanies syphilis, lepra and tuberculosis, so that mastoid conditions in luetics of fifty may resemble those normally deferred until three score and ten. Thin minor cellular divisions break down with great speed in the aged, and even the resistent sinus plate or tegmen may display extensive openings, without the warnings of rapid temperature fluctuation, increasing leukocytosis and polymorphonuclear percentage found under similar circumstances in the young and robust. This speedy advance, often with pale membrana tympani, is of course far more likely with the streptococcus mucosus.

Pain in the aged is very likely to be felt elsewhere than in the affected mastoid—referred to the vertex, in front of the ear, down the neck or to the forehead. Tenderness over the tip or antrum may not be marked at any time, owing to the hard cortex. The tympanic membrane may not bulge, may show little or no redness, and that only in Shrapnell's membrane, yet may pour out whitish or yellow pus under pressure when incised. If comparison be made with the sound side, dropping of the posterosuperior wall of the canal will be found not infrequently. Leukocytosis and temperature may not show much increase; such indices of bodily resistance are not raised by the old and enfeebled.

Preliminary radiographs have much confirmatory value regarding the destruction of bony trabeculæ in cases otherwise

obscure; but here, as at other ages, the ray must have clinical backing.

If the ordinary conservative measures prove ineffectual and operation becomes imperative, certain points demand respect:

1. Complete preparedness of everything and everybody concerned, before the anesthetic starts; shaving, skin, draperies, aprons, instruments, solutions, sutures, drainage, dressings, adhesive, anesthetist, nurses, surgeon and assistant. This commonplace is nowhere more essential than in dealing with old persons, who tolerate long delay on the operating table very badly. A warm room and a warm and properly padded table are not infrequently forgotten, to the dire discomfort of the patient.

2. Anesthesia should be the easiest possible; preceded by an opiate, usually well borne in combination with atropin; and in general gas-oxygen will be found best, with warmed ether vapor as an adjunct if strictly necessary. Danger from acidosis and renal irritation will forbid ether even more than the respiratory hazards. Local anesthesia, preferably by the technic of Graham,[8] following Neumann—injection of 2 per cent novocain, paying special attention to the external auditory meatus—may be used in imperative cases. Nevertheless, the psychologic risk from the needle pricks and from the grinding and crunching bone work must not be forgotten. White[9] reports a case, aged 81, started under local, in which ether had finally to be used.

3. Speed by the operative team means only a shortening of the shock and of the anesthetic risk; it must never be an end in itself. In no class of cases, however, must the surgeon be more certain of his way. Precious minutes ought not to be sacrificed tying surface vessels which an Allport retractor would hold; time must not be frittered away in the search for the antrum, even though old bone be brittle as glass and hard as adamant. Meticulous polishing is unnecessary in the cavities that are usually laid bare in operations on the aged.

4. The wound toilet and dressings must be rapid but thorough. Bone chips, suture ends, gauze frayings, are ill borne in aged structures with poor reparative power. Tight gauze packing is a rather barbarous infliction upon old people. An oily dressing, made with dichloramin—2 per cent—chlorcosane

dropped into the cavity and held there by a short strip of narrow gauze packing folded into the lower angle of the wound, as suggested by Fenton,[10] or injected through small soft rubber tubes in the upper and lower angles of the wound, according to Kistner,[11] is by far the most comfortable, as it is the swiftest dressing in the destruction of suppurative processes. Aqueous Carrel-Dakin solution may be similarly injected, as proposed by Mahu,[12] but has the disadvantage of requiring injection every two hours and of maceration of the skin. The latter difficulty may be controlled by vaselin or zinc ointment about the wound. Bacteriologic control of the wound, by daily counting several fields from the wound secretion, will permit suturing as soon as but one organism is found to a field. This refinement is not, however, a necessity with the oil dressings.

Erysipelas may readily occur from tight bandaging, poor circulation, abraded skin or chemical dermatitis. Usually mild, it may condition grave medical complications.

It must never be forgotten that the old person is far more a medical than a surgical risk; and the internist must be constantly on guard against hypostatic pneumonias, pleurisies and the cardiac and renal disorders incident to sudden septic infective processes in those of minimal resistance. Further, the effect of suggestion, in showing the old person as soon as may be that his hearing remains and is improving, is a valuable aid in his convalescence from mastoiditis.

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LVII.

# ALCOHOL INJECTIONS AS A POSSIBLE ADJUNCT TO TONSILLECTOMY UNDER LOCAL ANESTHESIA.*

By Robert Sonnenschein, M. D.,
Chicago.

The writer has always favored the use of a local anesthetic in tonsillectomies wherever it could possibly be used. The advantages of local anesthesia seem to lie in several facts. First, there is either no nausea or, if any, much less than there is with general anesthesia. Secondly, there is the avoidance of aspiration pneumonia. Third, the time of operation is usually considerably shorter than where a general anesthetic is used, particularly in adults. Fourth, there is usually less immediate discomfort in that the patient can take ice, fluids, sedatives, etc., shortly after the operation, whereas with ether it is necessary to deprive the patient of these things for a considerable length of time. Fifth, there is the avoidance of the ill effects that ether or chloroform may have upon the general system, such as cardiac depression, nephritis, or the induction of a coma in diabetes. Sixth, the swallowing of blood and the consequent vomiting are eliminated, in that the patient is able during a local anesthesia to expectorate whatever blood accumulates in the throat.

We have employed local anesthesia in tonsillectomies for many years in all cases above the age of 14 or 15, unless the individual was too apprehensive of the operation or the pharyngeal reflex uncontrollably active. No hypodermic was given before the operation unless the individual needed a sedative. We have often observed that when morphin was used the patient was often markedly nauseated thereby and was likely to vomit while being anesthetized or during the operation.

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*Read at meeting of American Laryngological, Rhinological and Otological Society at Atlantic City, May 10, 1923.

Means Employed.—Years ago we injected the mucosa about the tonsil with one-half to one per cent novocain, with or without the addition of adrenalin. During the war novocain was unobtainable at the hospital at which most of our work was done, so we employed apothesin in the same dilution and have ever since used it with great satisfaction in many hundreds of nose and throat operations. The only unfavorable effect noted a few times was a moderate cyanosis or some fainting on the part of the patient. The latter symptom, however, was largely psychic, since it occurred a number of times before the needle had penetrated the tissues. Before infiltrating the mucosa the pharynx was, in some of the patients, swabbed with a small amount of 10 per cent cocain to reduce the pharyngeal reflex and render entrance of the needle less disagreeable. The writer has for many years, when injecting the region of the posterior pillars, entered the needle on the posterior and not the anterior surface. Otherwise the swelling of the posterior pillars causes a distortion of the parts so that it is rather difficult to see where the tonsil ends and the posterior pillar begins; but if the injection is made on the posterior surface of the posterior pillar this does not occur. We do not recall ever having seen this point mentioned in the literature.

In 1909, Yankauer described a most excellent method of producing local anesthesia for tonsillectomy by blocking the posterior palatine nerve at its emergence from the posterior palatine foramen. He states: "The tonsils are supplied by the circumtonsillar plexus derived below from the tonsillar branches of the glossopharyngeal, and above from the middle and posterior palatine nerves. The glossopharyngeal is the smallest of the cranial nerves and its tonsillar branches are small; it is, moreover, a nerve of special rather than of general sensation, and its tonsillar branches may for practical purposes be neglected. The middle and posterior palatine nerves are derived from Meckel's ganglion of the fifth nerve, which is the principal nerve of sensation for the entire head. These nerves leave the sphenopalatine fossa through the accessory posterior palatine foramen, two small openings in the posterior part of the hard palate at the junction of the alveolar process with the horizontal part of the bone. These two nerves supply

not only the tonsil itself but the soft palate, both pillars and a part of the lateral pharyngeal wall. The two openings are very close together, but their location is not marked by any special landmarks on the surface of the mucosa of the mouth, so that the point for injection must be determined by arbitrary measures. There are two methods: First, if the last upper molar tooth is present begin at the posterior end of the margin of the gum of the last molar tooth and measure upward one centimeter—that is, at right angles to the free border of the teeth, and then one centimeter backward. Second, if the last molar tooth is absent, we first determine the location of the tip of the hamular process by placing the finger upon the soft palate; the process can be distinctly felt, as a sharp point of bone.

Then measure one centimeter in an oblique direction upward and forward. When the last molar is present, these two measurements usually meet at the same point; when they do not, an intermediate point is selected. As the mucosa in this region is situated about one centimeter from the bone, the direction in which the needle is inserted is important. A needle with a long shank is used and applied to the mucosa at the point stated, the shank lying across the tongue and in the angle of the mouth on the opposite side. In this position it is inserted until the point touches the bone, when about 1.5 centimeters of a 1 per cent solution of cocain are injected."

The writer tried this method a few times and then for one reason or another discontinued it until the papers of John A. Thompson appeared in 1917 and 1920. He states that "the anterior surface of the tonsils is supplied with sensation by the posterior or small palatine nerve, a branch from the sphenopalatine ganglion. It emerges with a small artery from a separate opening immediately behind the posterior palatine foramen." This lies just anterior to the posterior edge of the hard palate and medially to the last molar tooth. "If the teeth are missing or unerupted, the nerve lies just in front of and internal to the hamular process of the internal pterygoid plate of the sphenoid, which can always be felt through the soft palate. The glossopharyngeal nerve also sends branches to the tonsils." Prof. Knower of the Univervity of Cincinnati

collaborated with Thompson and made sections of the neck as the level of the lower third of the tonsils showing the relation of the glossopharyngeal vessels and the tonsils. The important structures which run through this region are surrounded by a loose connective tissue which is continuous from the pharyngeal wall outward and back in front of the deep prevertebral fascia, to surround the carotids, hypoglossus, superior sympathetic ganglion and plexus, vagus and glossopharyngeal nerves. In blocking nerves it is not necessary to inject the nerve itself, since surface contact is all that is needed. The connective tissue noted by Knower permits us to surround the glossopharyngeal nerve with anesthetic solutions without any danger of injuring the other important structures above mentioned. The injections can be made between the tonsils and anterior pillar, penetrating the constrictor muscles and pharyngeal fascia. A better route is external to the anterior pillar at the junction of the upper and middle thirds of the tonsil. At this point there is only mucous membrane to pierce, and there is no danger of passing the needle through a deep tonsillar crypt as in the first method and of carrying infection into other tissues with serious results. At this level the nerve trunk is three-quarters of an inch from the surface." Thompson and Tucker made a series of experiments to see if undesirable effects would result from surrounding other vessels and nerves of the neck with the anesthetic solution employed (namely, 1 per cent novocain with 10 per cent of adrenalin chlorid solution in the menstruum), after first making a surface application of 20 per cent cocain on the palate and pharynx. Their conclusions were that if the vagus on both sides was surrounded by the anesthetic solution or even a "direct injection of novocain solution was made into the nerve it had no harmful effect." After the preliminary surface application above mentioned Thompson injected 10 minims on each side of the palate and two drams external to each tonsil.

This method was adopted with most excellent results and has usually given us perfect anesthesia and as little bleeding as any other method we have ever attempted. We believe that the operative technic and bleeding are relative matters; whether a sharp instrument, a dull ring or any other appliance is

used, some cases bleed greatly and others very little. This seems to depend on several factors, such as the anatomic peculiarities of the parts, like the presence of rather large veins in unusual places, etc., the direction in which vessels are severed, the clotting time of the individual, the depth in the tissues to which an incision is carried, etc.

Because of its toxicity, the writer has never used cocain, even in weakest concentrations, for submucous injections in the nose or throat. In nasal operations we have applied it topically either in the form of flakes or a 10 or 20 per cent solution together with adrenalin chlorid. In tonsillectomies, as before mentioned, it has been used only by means of swabbing the mucosa of the pillars, palate and posterior pharyngeal walls preliminary to an injection of apothesin or novocain with adrenalin, in very apprehensive individuals or those with very active pharyngeal reflex. However, we have always felt that the less cocain employed the better.

A few weeks ago Dr. Ellison L. Ross read a paper at the April 5, 1923, meeting of the Chicago Laryngological and Otological Society on "Some General Effects of Local Anesthetics Administered as in Tonsillectomy," in which he showed that cocain, even when applied locally, sensitizes the individual to adrenalin so that the blood pressure rises greatly when the latter is injected. This synergistic action produces an increase which is much greater than the combined increase of blood pressure produced by cocain or adrenalin used separately. In animal experiments, this blood pressure increase was shown to be especially high as regards the intracranial venous pressure which increased to 467 per cent. This probably accounts for many of the unpleasant symptoms noted at times when cocain application is followed by the use of adrenalin. Dr. Ross states that "intracranial venous stasis produces cerebral and medullary asphyxia. The former would cause, first, a brief stimulation evidenced by anxiety, restlessness and irritableness. This would be followed by cortical depression which would lead to faintness. Medullary asphyxia would produce the dyspnea, rapid breathing and pallor."

In view of these facts caution should be observed in our anesthesia routine. Dr. Ross stated that if adrenalin be first

used and then a pause of five or ten minutes made until the blood pressure effect has disappeared, the application of cocain will not result in the greatly increased blood pressure noted where cocain is first applied.

Recalling the good results reported by neurologists and surgeons in getting at least temporary relief from pain in cases of neuralgia by injections of alcohol into the nerves or their immediate vicinity, it was deemed worthy of a trial in tonsillectomy. This procedure was comtemplated at first not so much for procuring perfect anesthesia during the operation, since we already had that by means of the nerve blocking with aposthesin or novocain, as outlined, but it was directed towards reducing or rather mitigating the very painful condition usually present for some days after the operation. Although aided by the American Institute of Medicine, we were unable to find references in the literature on the use of alcohol injections for this purpose, either into the posterior palatine nerve, the glossopharyngeal nerve or the pharynx itself. In order to be certain that the addition of alcohol to a solution containing apothesin and adrenalin would not cause an incompatibility with the production of deleterious substances, the research department of Parke, Davis & Co. was consulted. They assured us that "no chemical antagonism exists between either apothesin or adrenalin and alcohol." They considered it "inadvisable for such a mixture to remain standing more than a few days, since there is always a possibility of slight traces of aldehyde in the alcohol gradually affecting the adrenalin." "In the case of apothesin no indication of instability in the presence of alcohol was found, even after a longer time than a few days." Since we always mix the apothesin, alcohol and adrenalin just before injecting, there can be no danger of any changes occurring in the solution.

Regarding the effect of alcohol on the tissues, Otto May, after quoting Schlosser, Finkelnberg and others, concludes that (1) "Alcohol injected near the trunk of a peripheral nerve produces more or less complete necrosis at the point of injection. (2) This change is not ascending; the nerve above the place of injection remaining normal. The cells of origin (ganglion) may show some chromatolysis but no actual per-

manent injury. (3) The conditions produced by the injections are more favorable to regeneration than nerve sections without suture. The anatomic continuity of the nerve trunks favors rapid regeneration, although this may be somewhat retarded by the fibrosis which occurs to a greater or less extent in all alcohol injections." If alcohol was put merely around the nerve its effect, although marked, was transitory; the nerve quickly recovered its function. (Quoted by G. Sluder.) Probably the effect on the other tissues is also a local necrosis followed by a fibrosis.

As to the systemic effects of alcohol, most pharmacologists agree that even in small doses the action on the central nervous system is quite pronounced. It gives rise to a feeling of well being and the individual becomes more self-confident and less shy. Large amounts blunt the sensations. Binz's theory is that alcohol first stimulates the central nervous system and then depresses it. Schmiedeberg's theory is that alcohol depresses the central nervous system from the very beginning, picking out those centers first which were developed last; thus the finer degrees of judgment disappear first. With reference to its action on the circulatory system small doses of alcohol slightly accelerate the pulse and stimulate the heart. The vessels are not much affected, although there is a peripheral vasodilatation. The blood pressure usually rises somewhat.

Various concentrations of alcohol were used by us, from 25 per cent to 95 per cent, but it was found that from 33 to 50 per cent apparently served best in that it had a good effect without the marked reaction. The solution was prepared just before use by mixing about two and one-half drams (10 cc.) of a one-half of one per cent or a one per cent solution of aposthesin with one and one-half drams (6 cc.) of 95 per cent alcohol and adding thereto ten drops of a one to one-thousand adrenalin chlorid solution. In apprehensive persons the mucosa covering the posterior third of the hard palate and the posterior pharyngeal wall was swabbed two or three times with a very small amount of a 10 per cent cocain solution in order to render the entrance of the needle less painful and to diminish the pharyngeal reflex; but where this preliminary cocain application was entirely omitted the anesthesia was just

as good. A straight needle attached to a syringe of one dram (4 cc.) capacity was used. About five drops (0.3 cc.) were injected on each side just lateral to the last upper molar tooth just anterior to the posterior edge of the hard palate. About one or one and a half drams (4 to 6 cc.) were injected external to each tonsil into the areolar tissue at the level of the junction of the upper and middle third of the tonsil and a small amount, six or seven drops (0.5 cc.) on a level with the base of the tonsil. At times a few drops were also injected into the posterior surface of the posterior pillars. After the region of one tonsil was completely injected, the other one was likewise treated and then a pause of from five to eight minutes was made before beginning the removal of the first tonsil. Dissection with knife and snare was done in practically all these operations, which now number 92 private cases and a very considerable number of ward cases.

Results Obtained.—The anesthesia produced by the method outlined was very good. As previously stated in this paper, excellent results, however, had already been obtained by the use of apothesin alone. The advantages which we see from the addition of alcohol to the anesthetic solutions are several. First, there were practically no toxic effects produced from the anesthetic. This may possibly be due to the fact that less anesthetic solution was used than otherwise would have been the case, since one-third or at times even one-half of the fluid used for injection consisted of alcohol. The report of the Committee on Local Anesthesia of the Section on Ear, Nose and Throat of the American Medical Association, on which the writer had the honor of serving, showed that unfavorable symptoms were rarely caused by novocain and apothesin solutions when properly used. It seems reasonable to suppose that the dilution of the anesthetic by the addition of alcohol renders the probability of trouble more remote. Secondly, there was much less syncope. The class of patients which the writer had to operate upon included many very excitable and apprehensive individuals who ordinarly cause a surgeon much trouble in that they become very "nervous" and often "faint" or threaten to do so during the course of the operation itself. Since using the alcohol, however, we have had practically no trouble from this source. Thirdly, there has seemed to be con-

siderably less postoperative bleeding; hemorrhage is, however, a relative matter, and does not seem to depend upon the method of operation but upon other factors previously mentioned. Still, in this series of cases we did notice considerably less postoperative bleeding than usual. This may, however, have been merely coincidental and not consequential. Fourth, during the first 24 to 72 hours most of the patients seemed to have less discomfort than is usually noted. There were some, of course, who complained very bitterly and others who had practically no pain at all. The majority, however, seemed to have much less pain and dysphagia than usual. The committee on local anesthesia recommended that most operations be performed in the reclining or semireclining position, as there was less tendency to syncope, etc., but our cases remained in a sitting posture and there was no difficulty whatever from that standpoint.

While the above mentioned advantages seem to pertain to the use or addition of alcohol to the anesthetic solution, there were several unpleasant features which should be mentioned. The title of this paper gives alcohol as a possible adjunct in local anesthesia for tonsillectomy. It is necessary and important, therefore, to weigh the evidence in order to see whether the advantages far outweigh the disadvantages. Many operations must be performed before it can be definitely stated that alcohol deserves a permanent place in the rôle we have assigned to it. Some of the unpleasant features noted were that there was more pain at the time the fluid was injected than when only novocain or apothesin alone were used, but this pain is transitory. Second, there is always a possibility of some impaired motility of the soft palate for some time, due to a blocking up of the motor nerve. This effect was noted in several patients for a period of from ten to fourteen days, and in one individual there was a complete immobility which persisted for almost four weeks, with recovery after faradization. In the latter case some of the alcoholic anesthetic fluid had been injected to or rather behind the posterior pillar. For this reason we advise (1) omitting injection behind the posterior pillar when employing alcohol; and (2) would also suggest using no greater amount of anesthetic solution for infiltration of the two commonly selected areas (the regions

of the posterior palatine and glossopharyngeal nerves) than is needed to prevent pain. With the impairment of palatal mobility, not only is the speech affected, giving it a socalled nasal twang, but the swallowing of fluids is difficult and they often run out by way of the nose. In a few cases considerable swelling of the tissues was noted for several days, due probably to marked reaction to the alcohol injection. These effects have, however, been observed for some days postoperatively by most men who use infiltration methods of anesthesia, whether dilute cocain, novocain, apothesin or other solutions were employed. In spite of these symptoms, however, we believe that the addition of alcohol to anesthetic solutions deserves a thorough trial, since it apparently has some decided advantages as compared with novocain or apothesin alone.

## CONCLUSIONS.

Local anesthesia in tonsillectomies, wherever it can be employed, is preferable to general anesthesia in that it causes less nausea, less vomiting, and less immediate postoperative discomfort. The time of operation is usually shorter than with general anesthetic and there is no danger of aspiration pneumonia. Furthermore, the systemic effects of ether, chloroform, etc., such as nephritis, cardiac depression, the induction of coma in diabetes, etc., are avoided. The addition of alcohol to the anesthetic fluid seems to offer certain advantages in the way of less toxicity, less syncope and less postoperative discomfort and bleeding, which we believe far outweigh the transitory unpleasant symptoms, such as sharp pain at the time of injection and the possibility of temporary interference with the mobility of the palate.

To my associate, Dr. S. J. Pearlman, my thanks are due for much assistance with the operative and other work connected with the preparation of this paper.

Since reading this paper, the author has found that the injection of the anesthetic solution alone (without the addition of alcohol) into the posterior palatine nerve, and injecting as previously described the anesthetic solution containing from 25 to 33 per cent of alcohol into the region of the glossopharyn-

geal nerve, have given all the favorable effects above noted, without causing any interference with the movement of the soft palate after the operation.

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LVIII.

# THE CLINICAL DIAGNOSIS OF LARYNGEAL TUBERCULOSIS.*

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The purpose of the present paper is to bring before you one of the most frequent complications of tuberculosis, the laryngeal, and to plead for its recognition, with the hope that the realization of the necessity for early diagnosis will lead to the cure of many cases and the arrest of others.

It is hardly necessary for me to state that the type of laryngeal tuberculosis considered is secondary to pulmonary tuberculosis. Such a statement would be superfluous, were it not for the fact that occasionally one hears of the primary form. The primary type is so extremely rare, if indeed it exists at all, that it scarcely deserves serious consideration. Arrowsmith[1] states: "It is conceded today that primary laryngeal tuberculosis probably never occurs and that most individuals who exhibit tuberculous manifestations in mature life have been infected in childhood or infancy, the infection remaining latent as a rule, until some adventitious stimulus spurs it into activity in all of its protean forms." Wood,[2] however, believes that it is unquestionable that primary tuberculosis of the larynx has occurred, though it is exceedingly rare.

Many laryngologists of wide experience, such as Rr. Robert Levy, state that the primary type of the disease has almost never been encountered in their many years of experience in the diagnosis of laryngeal tuberculosis. Hastings[3] says: "Tuberculosis of the larynx is rarely, if ever, primary. The primary focus may be undemonstrable except by autopsy. In view of the numerous positive tuberculin test observations and confirmatory autopsy records in apparently nontuberculous

*Read before the Mid-Western Section Meeting of the American Laryngological, Rhinological and Otological Society at Omaha, Nebraska, February 21, 1923.

patients a diagnosis of primary tuberculosis of the larynx can well be questioned, certainly a verdict of 'not proven,' so far as its being primary is legitimate, until a searching autopsy is made." Levy made a similar statement in discussing Hurd's[4] paper. Sir St. Clair Thomson[5] states: "Primary tuberculosis of the larynx, as shown by postmortem integrity of the lungs, is so extremely rare that for practical purposes its possibility may be neglected. When cases are met with where no pulmonary lesion can be detected this is simply because our present methods of investigation are inadequate to detect early or limited deposits in the chest."

Inasmuch as the laryngeal complication is almost as frequent as the intestinal form in patients suffering from pulmonary tuberculosis, this subject deserves serious consideration from the entire medical profession. Because some of us live and practice in Colorado we can appreciate the seriousness of the problem confronting our profession on account of the large number of people who come to the West annually in search of health. That we should and do feel the responsibility more keenly than some others is only natural. Levy[6] has said: "No subject in medicine possesses a more extensive or interesting history than tuberculosis, nor can any disease boast of more attempts at solving the problem of its treatment with less permanent, definite and satisfactory results." Bane[7] states that vigorous efforts should be put forth to check the local disease. Dean[8] and Mullin[9] have shown the desirability of cooperation between the laryngologist and internist if the best results are to be obtained for the patient.

Brown[10] says that laryngeal tuberculosis occurs in about 25 per cent or more of adults with pulmonary tuberculosis, slightly more in men than in women, and next to tuberculous enteritis and colitis is the most frequent complication of pulmonary tuberculosis, due most likely to direct infection of the part with the sputum. "Even early cases—cases in the incipient or minimal stage—are not spared (12 per cent), but as the pulmonary disease progresses the laryngeal complication becomes more frequent (moderately advanced, 26 per cent; far advanced, 45 per cent). The significance, then, of a complication so frequently seen among patients with pulmonary

tuberculosis cannot be exaggerated." Thomson[5] gives very similar percentages in his textbook.

One of the very earliest symptoms about which a patient will complain is a slight huskiness of the voice. The laryngologist should be on the alert for the early symptoms. Minor,[11] Curtis,[12] Thompson,[13] Carmody,[14] Grayson[15] and many others have emphasized the importance of an early diagnosis. At first this slight huskiness may not be constant, but is present only upon getting up in the morning or after talking later in the day. As the disease progresses in the larynx the huskiness may become constant and may even amount to decided hoarseness. It has not been our experience that aphonia usually begins early in the disease and persists, except in the severe or rapidly fatal cases.

Some patients have so little involvement of the lungs that the laryngeal symptoms predominate, while others are not aware of any pulmonary disease. In fact, early cases may rarely heal spontaneously. Levy[16] has recently mentioned this. Some of the victims like to believe their cough and expectoration are due entirely to the laryngitis. In this belief patients may be sincere, but in some cases it is because they are hypersensitive mentally regarding their disease and are trying to conceal the pulmonary lesion. It is in just such mild cases as this that a very careful physical examination of the chest by a competent internist, supplemented by stereoscopic X-rays, will show the slight but positive involvement of the lungs even very early. That such early cases should, and frequently do, seek the advice of a laryngologist first is only to be expected. Hence we as laryngologists must be on the alert in the recognition of the earliest laryngeal manifestations clinically. Mullin[17] has urged greater laryngologic responsibility in such cases, because of the importance of an early diagnosis.

Solenberger[18] found "mere weakening of the voice, vocal fatigue on talking, hoarseness, a sense of dryness in the throat, troublesome localized tickling, a lumplike tickling on swallowing, pain, a feeling of languor or debility, and increased dry cough," all important in the early stage of the laryngeal disease. He mentioned also recurrent acute laryngitis as a frequent symptom. He stated: "These signs no doubt often

herald the approach of the disease and should always be an indication for speedy laryngoscopic examination. But they are also signs of actual involvement. Sometimes nearly all these external signs are absent in the process of involvement, even to the point of disintegration."

In moderately severe types the decided hoarseness or even aphonia with cough, expectoration, loss of weight, anemia, anorexia or sometimes dysphagia present an almost unmistakable picture to those experienced in the diagnosis and treatment of laryngeal tuberculosis. It follows, almost without saying, that with aphonia, dysphagia, anorexia, secondary anemia, emaciation, constant cough and expectoration, night sweats, fever, etc., the clinical picture is too evident almost before the larynx is examined. If this deplorable stage can be prevented by an early diagnosis a great deal of suffering and loss of human life can be avoided.

In a paper which the author[19] read before the American Academy of Ophthalmology and Oto-Laryngology in September 1922, the earliest sign of this disease was given as infiltration over the posterior pharyngeal wall. Dr. Robert Levy, in a personal communication, fittingly suggested that it had been his experience that vascular changes often occur earlier than infiltration. I have, therefore, included hyperemia and anemia as the earliest changes in the following classification:

1. Vascular Changes
   a. Anemia
   b. Hyperemia.
2. Infiltration
3. Tuberculoma
4. Ulceration
   a. Acute or Active
   b. Chronic or Sluggish
   c. Superficial
   d. Deep
5. Edema
6. Perichondritis
7. Chondritis
8. Necrosis

Infiltration often is first manifest at the interarytenoid sulcus, or below this point over the posterior laryngeal wall, and is due to sputum remaining in contact with the mucous membrane for hours at a time during sleep if we accept the older theory, advanced by Louis.[20] It is difficult to explain the early involvement of the posterior half of the larynx in any other way. Minor[21] has recently shown many of the early manifestations involving the posterior half of the larynx. Infection through the lymph and blood stream, as advocated by Heinze, is just as probable. If the infiltration becomes more extensive the lateral walls, the arytenoids, the true and false cords, or even the epiglottis may be involved. Thomson[5] states that different parts of the larynx are usually involved in the following order: "(1) The arytenoids, (2) the interarytenoid region, (3) the vocal cords, (4) the ventricular bands, (5) the epiglottis." Kyle[22] says: "The epiglottis is a favorite site for tubercular infiltration, and this organ may assume a simple globular, puffed form, a thickened crescentic shape, or simulate the Turkish turban—the socalled 'turban' epiglottis." He mentions also the importance of the "club shaped" arytenoids when the latter are infiltrated.

Slight elevations above the surface, due to tubercles or tuberculomata, may occur with the infiltration or may follow the infiltrative stage. These may have a smooth exterior, but are usually uneven and resemble a papilloma. In fact, a tuberculoma is frequently mistaken for a papilloma. Trautman[23] has stated that tuberculoma is among the earliest manifestations.

Tuberculous ulcers in the early stages are superficial and have "mouse eaten or mouse nibbled edges." While they may be acute or active they are far more often chronic or sluggish. They are usually covered with a thin layer of sputum or pus and are superficial. Later in the disease the ulcers may be deep, but they do not have the sharply defined "punched out" edges so often seen in lues. The above clinical picture is by no means always so typical, especially when lues and tuberculosis exist together. Carmody[14] states that where there is much scar tissue we may and do frequently have syphilitic infection also. Grunwald[24] has emphasized the importance of watching

for lesions in the larynx due to syphilis and tuberculosis. He shows colored plates in his text which illustrate the dual infections. Mixed infections, which are so common in pulmonary tuberculosis, also mask the objective signs and symptoms. Ulcers are common over the vocal cords but rare over the arytenoids. The ventricular bands and epiglottis are often ulcerated too.

Fetterolf[25] has classified the ulcerative stages as follows:

(a) Infiltration with superficial ulceration, (b) infiltration and deep ulceration, and (c) infiltration with superficial and deep ulceration. This classification has much in its favor, because we know that frequently the ulcers form on an infiltrated base.

Edema is so often designated as "ashen gray" when seen in laryngeal tuberculosis, and while it usually occurs late, it may be present early. Personally I always feel less optimistic regarding the prognosis when there is extensive edema of the larynx. The mucous membrane covering the edematous areas looks smooth and tense. The blood supply is sooner or later seriously interfered with because of the constriction of the blood and lymph vessels. The landmarks are obliterated by the edema very much as they are in the nose by hyperplastic rhinitis and ethmoiditis. In the larynx it is rarely a true edema, but it narrows the lumen of the larynx. Lockard's[26] textbook illustrates this well and his[27] more recent article is well worth reading.

Edema may be preceded by perichondritis. However, the edema often hides a perichondritis or chondritis, and it is by no means easy, or sometimes possible, to say in a given case which type of clinical pathology occurred first. It is almost needless to emphasize that while a clinical classification is important, not all cases are of the textbook type. Syphilis may and often does produce a laryngeal picture not at all unlike that of tuberculosis. Thomson[5] says: "Perichondritis of the thyroid may produce a swelling of the ventricular band, if the inner surface is attacked. The purulent collection makes its way into the larynx. The external surface of the thyroid plate may also be affected. A swelling then takes place externally which is not inflamed and is seldom painful. An incision into it yields pus and leads down to denuded cartilage."

If the patient lives long enough there is apt to result from the edema necrosis of the cartilages of the larynx with severe dysphagia and a fatal termination. The necrosis of the cartilages may take place slowly but is usually rapid. Thus late in the disease the patient is usually emaciated from the prolonged illness, fever and difficulty in swallowing. Lockard[28] has shown microscopically that the necrosis of the cartilage of the epiglottis may be due entirely to tubercle bacilli and that it is not necessarily dependent upon a secondary mixed infection.

In conclusion permit me to state that while an early diagnosis does not always mean a complete recovery, we do know that 70 to 80 per cent of our cases of pulmonary tuberculosis are curable if the diagnosis can be made early and proper treatment begun. Thompson[13] states in speaking of laryngeal tuberculosis that progress is ready to hand in the making of an earlier diagnosis of local infection. A laryngeal complication serves only to lessen a patient's chances for recovery and often prolongs the time required for an ultimate arrest or cure of the disease. Freudenthal[29] believes "The prognosis of an established tuberculosis of the upper air passages is better than it used to be years ago." Thomson[30] states that "tuberculosis is one of the most common and most deadly scourges of humanity. There is no other which slays so many of our people in the very prime of their career, for it causes one-third of the total mortality during the chief working years of life. It kills 53,000 individuals annually in the small population of England and Wales. It is the greatest cause of disablement in adult life. It leads to more loss to the family and to the nation than any other single disease. It is one of the saddest afflictions."

Freudenthal[29] has advocated that "all patients suffering from pulmonary tuberculosis should be advised to undergo a laryngologic examination, not only when the disease is diagnosed or upon their entering a sanitorium, but also at regular intervals, irrespective of their complaints." Mullin[17] has urged such an examination. Certainly it behooves all of us as laryngologists to be on the alert for the early manifestations if we are to succeed in curing tuberculosis.

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PHYSICIANS' BUILDING.

LIX.

# THE DIFFERENTIATION OF NEUROLABYRINTHITIS SYPHILITICA AND LABYRINTHITIS SYPHILITICA TARDA.

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These two forms of syphilis of the hearing apparatus are entirely different affections occurring at widely different stages of the same general disease. They present different pathologic and clinical aspects and prognoses. Furthermore, they may be found eventually to require different forms of treatment, judging from the different results obtainable from the same form of treatment.

As early as 1907 and 1908, while studying the galvanic reactions of a large series of deafmutes, it was observed that the majority of cases of deafness of the socalled nerve type presented, as might be expected, a negative galvanic reaction of one or both branches of the eighth nerve. These observations covered cases of nerve deafness from many different causes. Prominent in the list of causes, particularly among those developing deafness after puberty, was to be noted syphilis, and the majority of these presented negative electrical reactions of the nerve.

The reactibility of the vestibular branch of the eighth nerve was interpreted as negative when neither the positive nor the negative electrode applied to the external auditory canal, with a current strength of 20 m. a., or as much as the patient could tolerate, produced the slightest alteration in an existing nystagmus or caused a nystagmus to appear that did not exist before.

A corroborative test was generally employed, that of opening and closing the current, using first the cathode and then the anode with a current strength of 6 m. a. If closing the cathode or opening the anode produced no momentary arrest of an existing nystagmus to the opposite side nor caused one

to appear to the same side, the vestibular branch of the eighth nerve was considered nonreactive.

The reactibility of the cochlear branch of the eighth nerve was tested after the same manner as the vestibular; however, the reaction when positive is different. The reactibility of the cochlear branch is indicated by a subjective noise, tinnitus; for instance, the cathode produces in the case of a reactive nerve a distinct tinnitus or increase of an existing tinnitus with a current strength of 4 to 6 m. a. When no such reaction is obtainable with 20 m. a., we may conclude that the nerve is nonreactive, nonconductive; in other words, out of function. In this particular type of case it would be superfluous to try the anodal reaction.

Normally with 6 m. a., closing the cathodal current or opening the anodal produces a momentary, but distinct booming sound in the ear of the side to which the electrode is applied. When no such sound is produced it is accepted as evidence of a negative reactibility of the nerve. A stronger current may be tried if one does not feel satisfied with the test made with 6 m. a.

The reactibility of both cochlear and vestibular branches of the eighth nerve was observed to be negative in the majority of cases of deafness presenting the history and signs of syphilis. Cases were found now and then among the syphilitic group which, in spite of complete deafness and nonreactibility of the vestibular labyrinth to the turning and caloric tests, presented quite normal electrical reactions. This seemed so odd to us at the time that my collaborator, Dr. Alice V. Mackenzie, was prompted to publish these first two cases in the Wiener Med. Wochenschrift. These first two cases, by the way, happened in the clinic on the same day within a few minutes of each other. These experiences, which were so contrary to those we were accustomed to seeing, rather upset our previous calculations and made me, at least, feel that, after all, perhaps Barany was right as he practically expressed it in his now memorable monograph "that the galvanic test had no clinical value." At that time we could not, nor did we, attempt to explain these apparently exceptional cases presenting what then seemed to be freak reactions. We were content merely to publish them.

It was several years before the explanation dawned on us. Since 1907 we have seen many cases of both kinds of deafness of syphilitic origin, namely, those with negative and those with positive electrical reactions. The explanation is simple enough now. On looking back, the chief wonder is that it did not occur to us long before. The galvanic test had been serving us faithfully in differentiating neurolabyrinthitis syphilitica and labyrinthitis syphilitica tarda without our being aware of the fact prior to five years ago, but in a manner definite to our purpose since. What all this has to do with our subject will be unfolded in the course of the paper.

Let us proceed to discuss in their order the points of differentiation made in the opening paragraph of the paper. The first mentioned was that "these two forms of syphilis of the hearing apparatus are entirely different affections occurring at widely different stages of the same general disease."

Neurolabyrinthitis syphilitica, as the name implies, is a syphilitic inflammation of the eighth nerve and the labyrinth. To be more accurate anatomically, the condition could be better labeled meningoneurolabyrinthitis, for the neuritis of the eighth nerve is nothing more than the extension of a primary syphilitic meningitis. If there were no meningitis, there would be no neuritis. Acoustic nerve neuritis results from the extension of meningitis and, compared with the other cranial nerves, we find it the most common form of extension. Syphilitic acoustic nerve neuritis is found much oftener than syphilitic optic neuritis. Whether syphilitic meningitis ever occurs without some involvement of the eighth nerve is doubtful. Judging from the accumulated clinical data of Oscar Beck, Willcutt and the writer, one is led to believe that the majority of cases of secondary syphilis reveals evidences of syphilitic meningitis together with involvement of the eighth nerve. Furthermore, the more pronounced the meningitis is, the more pronounced the neuritis. However, the syphilitic infiltration of the eighth nerve is rarely, if ever, quite so pronounced as the infiltration of the meninges. The pathologic findings of Asai, Hofer and O. Mayer show a gradual diminution of the syphilitic infiltration of the nerve from its origin toward its periphery. In some cases, where the patient succumbs to the virus, the infiltration of the nerve barely reaches the

spiral ganglion, in other cases the infiltration reaches to the labyrinthine spaces. The involvement of the labyrinthine spaces has invariably been found less intensive than that of the nerve stem. In short, the meninges, particularly the soft membranes, are found to be involved to the superlative degree, the nerve to the comparative and the labyrinthine spaces barely positive in the average case. The greatest amount of material for microscopically pathologic study of aural syphilis has comprised the temporal bones of syphilitic babes, because they afford the most excellent material for the purpose. None of the pathologic researchers who have studied the temporal bones of stillborn syphilitic babes or those dying within a few hours after birth have yet been able to find one in which the eighth nerve has escaped involvement. This shows as plainly as can be the vulnerability of the eighth nerve to the virus of syphilis and corroborates completely the clinical findings of the gentlemen mentioned earlier. The hereditary form of syphilis that is fatal to infants before or shortly after birth is typically generalized syphilis, showing all the characteristics of a virulent secondary syphilis of the acquired form.

I have referred to the second form of aural syphilis as labyrinthitis syphilitica tarda to distinguish it from neurolabyrinthitis syphilitica. The object of using these two different terminologies is for the purpose of contrasting the two conditions from the pathologicoanatomic standpoint. In this second form of aural syphilis the labyrinth is primarily involved, and from every indication available at this time the nerve escapes entirely. In the first mentioned condition the nerve is definitely involved and the labyrinth but slightly. In the second, the labyrinth is definitely involved and the nerve apparently not at all. It is clear then that these two conditions, in spite of the fact that they both have a common origin, are "entirely different affections."

Contrasting these two conditions further, we find neurolabyrinthitis occurring during the secondary stage of syphilis, when the virus is widely disseminated throughout the body, a matter of a few weeks or months following infection. It, therefore, occurs early. On the other hand, labyrinthitis syphilitica tarda occurs very late, usually in the hereditary form of syphilis, when it forms one of the symptoms of the complex

known as Hutchinson's triad. It may occur also very late in the acquired form, but it is rather an unusual manifestation of acquired syphilis, while in the hereditary form it may be considered fairly frequent. According to Hutchinson, it never occurs before the fifth year, more often at about the age of puberty, and is rarely, if ever, seen after the thirtieth year. Notwithstanding, the writer saw and reported a case a few years ago of labyrinthitis syphilitica tarda in a congenital syphilitic that had begun when the patient was forty-three years of age. We see, therefore, that these conditions are not only "entirely different affections," from the pathologicoanatomic viewpoint, but they also occur at "widely different stages of the same general disease."

These two conditions "present different pathologic" aspects, as already pointed out. They may be compared with two separate syphilitic conditions; for instance, optic neuritis or neuroretinitis of secondary syphilis and interstitial keratitis, which occurs only very late in syphilis, more often in the hereditary form. Both of these eye conditions can lead to blindness, and both are due to the "same general disease," but in no respect may they be considered as the same pathologicoanatomic entity. Both neurolabyrinthitis syphilitica and neuroretinitis syphilitica occur early in syphilis and occasionally nearly or quite simultaneously. On the other hand, labyrinthitis syphilitica tarda and interstitial keratitis occur very late and often together or shortly following one another. Other points of comparison existing between neurolabyrinthitis syphilitica and neuroretinitis syphilitica appear farther on in the paper as well, too, as the comparison between labyrinthitis syphilitica tarda and interstitial keratitis syphilitica.

The clinical aspects of neurolabyrinthitis syphilitica and labyrinthitis syphilitica tarda differ sufficiently to permit of a ready differentiation.

In the case of neuritis of the eighth nerve, if the involvement is sufficient to impair the normal conducting power of the nerve, the impairment can be detected by the galvanic test conducted after the manner already described. In the case of labyrinthitis, on the other hand, with an intact nerve, the galvanic reaction remains quite normal. The writer has observed many cases of bilateral labyrinthitis syphilitica tarda

with complete loss of vestibular function to the turning and the caloric tests, manifesting perfectly normal reactions to galvanism. A classical case of the kind was shown and reported with minute details a little more than a year ago, before the Philadelphia Laryngological Society, by two students of the Graduate School in Philadelphia. Many cases of the kind have been seen by the writer, both before and since that time. Again, cases of complete bilateral deafness have been seen in which the galvanic reaction of the cochlear branch of the eighth nerve has remained normal. Other cases of bilateral loss, both of the hearing and the vestibular functions, from labyrinthitis syphilitica tarda have been examined with perfectly normal galvanic reactions of both acoustic and vestibular branches of the eighth nerve; so that from the standpoint of clinical diagnosis it is not at all a difficult task for the examiner to distinguish these two processes.

In many other respects these two forms of aural syphilis resemble one another. Both are due to the same general disease, hence the history and findings of syphilis would not be sufficient to solve the question. If, however, the case is one of hereditary syphilis, there is a very strong suspicion that the resulting deafness and impairment of vestibular function are due to labyrinthitis syphilitica tarda and not to neurolabyrinthitis syphilitica. If the case can be proved to be one of acquired syphilis, the suspicion rests more strongly on neurolabyrinthitis.

Cases are met with now and then in which the history and findings point strongly to generalized secondary syphilis but not strongly enough to be absolutely dependable. In this type of case the character of the galvanic findings, together with the functional hearing tests, even in the absence of symptoms referable to the parts, has frequently led to the positive diagnosis of syphilis, to be confirmed later by that most dependable of all, the therapeutic test. The experience of the writer prompts him at this time to the decision that if he were allowed the privilege of selecting but one of two alternatives in arriving at a diagnosis of syphilis, he would unhesitatingly decide in favor of the functional tests in preference to the Wassermann. Again, there are cases of only moderate impairment of function, vestibular or cochlear, and cases of begin-

ning impairment, in which the galvanic and functional hearing tests have given advance information, permitting thereby the institution of treatment earlier than might have been possible otherwise.

In neurolabyrinthitis we have, as a rule, bilateral impairment of hearing, which is progressive from the start, but it rarely, if ever, goes on to complete deafness. In labyrinthitis syphilitica tarda the onset is more abrupt (apoplectiform in some cases) than in neurolabyrinthitis and frequently goes on to complete deafness.

Neurolabyrinthitis syphilitica presents a rather favorable prognosis in those cases in which treatment is begun early and is vigorously pushed. Labyrinthitis syphilitica tarda, on the contrary, presents a less favorable prognosis, even when the treatment is begun within a few days of the onset. In the latter condition there is frequently a pronounced improvement following the initiation of treatment, but there is a tendency to relapse in spite of the treatment. Such relapses are common in labyrinthitis; exceptional in neurolabyrinthitis. Why this is, the writer is unable to answer. But it suggests to us that the same form of treatment is not as definitely indicated in both of these conditions any more so than the same antiluetic treatment will answer equally well for deep interstitial keratitis and neuroretinitis syphilitica. In neurolabyrinthitis syphilitica and neuroretinitis syphilitica antiluetic treatment, particularly mercury, accomplishes apparently wonderful results. On the other hand, in labyrinthitis syphilitica tarda and in deep interstitial keratitis this same kind of treatment occasionally works with some satisfaction, but more often it does not, while a few cases are apparently aggravated by the treatment. It is common knowledge on the part of ophthalmologists that the second eye frequently becomes involved with interstitial keratitis while the first is still under treatment. The results of treatment in labyrinthitis syphilitica tarda are quite as unsatisfactory as they are in interstitial keratitis. It is possible that the best form of treatment for these last mentioned conditions has not yet been found. It is evident from the foregoing remarks that the two forms of aural syphilis referred to in the paper present not only "different pathologic and clinical aspects and prognoses," but in addition "they

may eventually be found to require different forms of treatment, judging from the different results obtainable from the same form of treatment."

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1724 SPRUCE ST.

LX.

# PAROTID FISTULÆ AFTER MASTOID OPERATION.

By W. E. Grove, M. D., F. A. C. S.,

Milwaukee.

The appearance of parotid fistulæ after mastoid operations has been reported several times in the literature, but inasmuch as these reported cases have been very few, it was thought of interest to bring this subject to the attention of this society.

In 1906, when Kretschman reported his first case, he called attention to the absence of any notations concerning them in the literature. He reported two cases. One was a young man of 25, in whom an extensive resection of the entire mastoid process was necessitated. There was a postauricular incision 12 c. m. long, which granulated normally with the usual amount of wound secretion. The main portion of this wound healed in normal time, but he noticed one point at which there was a moon shaped opening, from which a clear watery yellow secretion exuded. This opening was at about the level of the mastoid tip. A sound introduced into it ran about 2 c. m. toward the inferior canal wall.

His second case was a child of 10. The entire mastoid wound healed promptly in eight weeks. Three and a half years later this patient came back, stating that six months before, following a blow on this ear, the wound became moist and had so persisted. In the postauricular scar at the level of the mastoid tip there was a fistula from which a thin limpid fluid exuded during the act of chewing. A sound introduced into it passed about 2 c. m. toward the inferior canal wall.

Combier reported three cases which occurred one and one-half, three and four months after the cure of the mastoid wound. They were all found at the lowest portion of the mastoid wound. Probes could always be passed about 2 c. m. toward the inferior canal wall.

Binnerts reports a case in a child of 5, where a bilateral mastoid operation had been done five months before by another surgeon. Whenever the child chewed or moved the

jaw a fluid appeared from a fistula situated in the lower part of the right scar. There was no similar trouble on the left side.

In addition to these cases, two more were reported by Claus, three from Passow's clinic, and one by Marres of Amsterdam. Cases were also reported by F. C. Schreiber, Philip Hammond and C. Canesteo.

CASE REPORT.

On March 3, 1920, there was admitted to Columbia Hospital a child, aged 8, with an acute mastoid on the right side. This followed an attack of influenza two weeks before. There had been a severe earache three days before, when a paracentesis was done. Following this there was a copious discharge of pus from the ear.

Past history: Earache with every attack of sore throat. Has had several previous attacks of otitis media. Tonsils and adenoids were removed December, 1917. Remainder of the past history negative except for an attack of pneumonia at 18 months.

Physical examination: Brown purulent discharge from right external canal. Tenderness over right mastoid region but no edema. Glands of right side of neck are slightly enlarged. Left ear negative. Chest negative. Abdomen negative. Reflexes all present, but those of extremities are exaggerated. On admission, temperature 103. Pulse 102. Respirations, 22. Blood examination, 10,850 W. B. C. Differential count shows S. M. 4 per cent; L. M., 2 per cent; polys., 85 per cent; eosin., 2 per cent; trans., 6 per cent; baso., 1 per cent.

Operation, March 4, 1920: Typical simple mastoid operation done with a straight vertical postauricular incision extending from the level of the upper attachment of the auricle to the mastoid tip. The cortex was extremely thin. When this was removed extremely large mastoid cells were encountered, which occupied the entire mastoid process down to the extreme tip, and which were everywhere filled with a thin straw colored fluid. Culture from the wound gave Gram + coccus. Blood culture was negative. Wound was packed with an iodoform drain.

March 8, 1920. Marked nasal discharge for last few days.

March 12, 1920. Right dacryocystitis appearing with severe inflammatory signs.

March 16, 1920. Under gas anesthesia the abscess of the right lacrimal sac was incised and a small drain inserted.

March 28, 1920. Patient left hospital with both mastoid and lacrimal wounds in good condition.

May 4, 1920. Patient entered Mt. Sinai Hospital because of a rise of temperature, which had begun one week before. At this time the drum, which had closed, was reincised. There was not much discharge after the paracentesis, but there was swelling and tenderness in the region around the mastoid wound. Under gas anesthesia the mastoid wound was cleaned out and some infected cells in the zygomatic region not reached by the original operation were cleaned out. The elevated temperature fell to normal in three to four days, and the patient left the hosiptal on May 16, 1920. Between May 26 and May 31 she again reentered the hospital for observation because of a rise of temperature, but this gradually subsided.

The mastoid wound now gradually filled in until it was closed and the patient left town. While away she noticed that the wound became moist whenever she chewed or yawned. When she returned to my observation I made out a small fistula in the lower portion of the mastoid wound leading in the direction of the inferior portion of the auditory canal, and from which a thin limpid secretion was seen to issue whenever she chewed. Various attempts to close this by means of fused silver nitrate but with no success.

In February, 1922, during an attack of scarlet fever an extensive mastoid operation on the left side was necessary. The same very pneumatic mastoid was encountered as upon the right side. The entire mastoid consisted of very large cells extending to the extreme tip, into the zygomatic region and back into the diploic spaces. These cells were all involved in the process and were cleaned out. The lateral sinus was laid bare throughout a large extent because of a perisinus infection but was not opened. During this illness the right mastoid wound broke down, following an acute otitis media, and it was again opened and drained. The mastoid wounds both filled in very slowly because of the lowered resistance of the patient. After they had closed entirely a very tiny fis-

tula appeared at the lower portion of the left mastoid wound similar to that which had occurred two years previously on the other side. As far as I know, this is the only reported case of a bilateral parotid fistula following mastoid operations.

I have not been able to close the fistulæ in his case by silver cautery, but as time passes they are gradually closing themselves down so that at the present time very little fluid escapes from them. I have advised resection of the fistula and suture, but this has been refused by the patient.

All of these parotid fistulæ have occurred either during the closing of the mastoid wound or very shortly thereafter, with the exception of one of Kretschman's cases, wherein the fistula occurred three and one-half years after the operation. Binnerts believes that they are due to an injury to the parotid gland by a sharp instrument during the operation, but inasmuch as they occur so rarely there must also be some other cooperating factors, such as abnormally large parotid glands extending backward over the mastoid process. This may occasionally occur, according to Graupner, who also found accessory parotid glands on the surface of the mastoid. Another cooperating factor, according to Binnerts, is the curved incision affected by some surgeons for cosmetic reasons. Kretschman also calls attention to the fact that previous infections or inflammations in the parotid may increase its size and push it back into the operative field of a mastoid, where it could then be injured by a separation of the attachment fibers of the sternocleidomastoid muscle.

Another factor, which has occurred to me, is that the gland may enlarge because of infection in its neighborhood at the time of the mastoid itself.

In those fistulæ which develop late Combier is of the opinion that the actual damage to the gland is done at the time of operation; that this area then apparently heals with the formation of a retention cyst, which later breaks down, due to some insult or infection.

As to the treatment of these parotid fistulæ, Binnerts cauterized the fistula in his case with chromic acid but without success. Kretschman cauterized frequently with fused silver and cured both of his patients after having failed by curetting and cauterizing with the galvantic cautery. Passow was not

able to cure his patients in this manner. He then excised the fistula and sutured the edges. Combier cured his patients by injecting a few drops of tincture of iodin into the sinus. Hammond also excised the fistula and sutured. We must not forget that these sinuses may also close spontaneously.

This report would be incomplete without calling attention to a paper by Kretchman, published in 1914, in which he calls attention to the occurrence of parotid fistulæ in the external auditory canal and reports one such case of his own and one of Politzer. Inasmuch as the external canal and the parotid gland lie in such intimate connection it is conceivable that furuncles of the former and abscesses of the latter may form a fistulous connection between them.

In his own case, after cleansing the external canal, he discovered a bleb about 4 m. m. anterior to the drum, and at the juncture of the upper and anterior canal walls, which became more prominent on chewing. An examination of the secretion obtained from this region was pronounced by Abderhalben to be saliva. Politzer's case was similar, except that the fistula was at the juncture of the anterior and inferior canal walls.

Kretchman was of the opinion that there may have been some defects in the formation of the external canal, in which defects some parotid tissue came into close connection with the external canal wall. In both of these cases the middle ear and the sound perceiving apparatus were normal.

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## LXI.

# LYMPHOID TISSUE IN THE TONSILLAR FOSSÆ FOLLOWING TONSILLECTOMY.

By B. B. Grichter, M. D., and S. J. Pearlman, M. D.,

Chicago.

The recurrence of lymphoid tissue in the tonsillar fossæ following apparently complete tonsillectomy is of perennial interest to the surgeon. Pierce[1] divides these recurrences in two groups; one in which the lymphoid tissue at the base of the tongue is drawn up by a contraction of scar tissue in the capsule, and another in which there is a globular mass unconnected with any kind of tissue at the base of the tongue. The appearance of the last named type of lymphoid reformations in tonsillar fossæ, following apparently complete tonsillectomy by competent operators, can, we believe, be explained by a consideration of the development and anatomy of the tonsillar capsule, plicæ and fossæ. This explanation is confirmed by histologic findings in tissue removed from the plicæ and supratonsillar fossæ following tonsillectomy.

The tonsil itself originates in the third month of embryologic development from an eminence, the tonsillar tubercle (Hammar[2]) in a recess formed from the lower portion of the second pharyngeal pouch. The walls of this recess originate from the second and third visceral arches, which become respectively the anterior and posterior pillars.

The plica triangularis or anterior tonsillar fold springs from the entire length of the anterior pillar and tends to cover the tonsillar recess or sinus like a lid. From the posterior pillar a similar fold, the posterior tonsillar plica, still further narrows the entrance to the sinus. This last structure is, however, not constant.

From the mucosa of the sinus solid epithelial buds or sprouts invade the underlying fibrous tissue. This takes place about the fifth month of embryonal life. These buds may be single or branched. The central cells degenerate and are expelled

as horny plugs, leaving blind crypts which become compressed by the simultaneous development of lymphoid tissue.

The latter originates in the more superficial portions of the tunica propria of the mucosa. The deeper layer of the tunica propria is pushed outward and becomes the capsule. "The capsule covers all surfaces of the tonsil not covered by epithelium. Theoretically it includes only that part of the deeper layer of the fibrous mucosa of the sinus that covers the attached surface of the main bud of the tonsil. As the mucosa of the more internal surfaces of the sinus, including the plicæ, have lymphoid nodules developed in them, often directly continuous at the root of the tonsil with the lymphoid tissue of the main mass, it seems best to consider the capsule as including the fibrous mucosa of the entire sinus, even to the internal rim of the plicæ." (Barnes.[3])

"As the lymphoid mass grows it fills more and more completely the tonsillar recess and "finally bursts through the faucial opening of the sinus, the walls of which surround it, much as the calyx surrounds the growing bud. The plicæ, partly because it may be pushed forward by the tonsil and partly because of the forward growth of the tongue, may undergo a greater or less reduction at its inferior insertion. If no reduction takes place the tonsil is suspended as by a sling by the plicæ, and a deep fossa exists between the two anteriorly and inferiorly. Between this condition and one in which only a slight fringe of plica remains anteriorly all gradations may be seen. If a plicæ retrotonsillaris is present, there is a posterior fossa between it and the tonsil which is continuous with the inferior fossa if one exists. The superior lobe of the tonsil expands upward into the cul de sac which forms the superior angle of the sinus, the walls of which surround it like a hood. Between the two an extensive fossa remains which may be quite open or its epithelial surface may be in apposition. This is the supratonsillar fossa. Its size varies with the size of the original pocket and also with the height on the outer wall of the sinus to which the root of the tonsil extends. Its faucial opening is more or less covered by the upper part of the plica so that internally it is continuous with the anterior fossa and, if one exist, with the posterior fossa. The ventral walls of the sinus surrounding the

tonsil have a small amount of lymphoid tissue developed in them, sometimes with shallow crypt formation. This is especially true of the supratonsillar fossa and of the plica anteriorly. This lymphoid tissue is continuous at the tonsillar root with that of the main bud and must be considered as forming part of it. At times the amount of lymphoid tissue developed in the deeper layer of the plica is so great as to form a thick wedge shaped mass, which is continuous with the main bud of the tonsil at its base. Under these conditions the plica has the appearance of being directly attached to the tonsil, and the anterior fossa is converted into a deep vertical crypt. This is the socalled attached plica (Fetterolf)." (Barnes.[4])

The tissue studied in this work was obtained from children aged from four to fourteen, at the Michael Reese Tonsil and Adenoid Clinic. The tonsils were of varying degrees of hyperplasia, but with no evidence of recent acute inflammation. They were removed under general anesthesia with the Beck-Mueller tonsillotome. This instrument is the one in general and favorite use at this clinic and, as will be shown later, bears no relation to the recurrences. The excised tonsil was first carefully examined and the presence of the intact capsule noted before a specimen was taken from its fossa. Specimens were not taken unless the tonsil was considered successfully removed in one maneuver. Tissue was removed by means of a small punch forceps as near as possible to the anterior pillar where it forms an arch with the posterior pillar; better seen when the former is put on the stretch. No tissue was removed from the region of the lingual tonsil.

Altogether 100 operated fossæ were studied. Serial sections were made and every tenth section was analyzed. In fifteen instances (15 per cent) lymphoid tissue with distinct follicle formation was observed. In some cases shallow crypt formation was present. In ten additional specimens (10 per cent) lymphoid tissue of a diffuse variety was seen. Much of this was microscopic in character. In all, 25 per cent of operated tonsillar cavities exhibited lymphoid tissue. This is probably a minimum figure, because at times only one piece of tissue was removed. Had serial sections been studied, lymphoid tissue would probably be found in a larger number of instances.

We do not conclude that this extratonsillar but intracapsular lymphoid tissue is always responsible for these recurrences. This tissue, however, seems to be the only one from which such recurrences can take place and best explains the reformation of lymphoid masses high up in the fossa which occur now and then at the hands of competent surgeons using the best possible technic. It is true that demonstrable recurrence may not occur in all instances in which lymphoid tissue can be shown to be microscopically present, since factors as yet unknown and indefinable may determine recurrence in one instance and prevent it in another.

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25 E. Washington St.

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LXII.

# CLINICAL AND PATHOLOGIC STUDY OF TONSIL CASES.

By Austin G. Morris, M. D.,

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The general impression among laryngologists relative to tonsil surgery has changed markedly during the past few years and has assumed a more rational working basis upon which to draw accurate conclusions.

It will be remembered that for many years the custom and practice was to remove obstruction, whether found in the throat or nose. If found in the throat a slicing operation was performed for the removal of the projecting part, and in the nose the removal was made of whatever structure presented the least difficulty. Should the trouble be found in the ears the patient was sure to be subjected to the old reliable treatment, namely, inflation. The result of some of this work has, as might be assumed, been most unsatisfactory. It has in a general way made the patient, treated in past years, a permanent patient. It not only failed to give relief, or at the most only temporarily so, but by the surgical work being misdirected made sufferers who were constantly exploring new fields in hopes of gaining relief. To our own leaders and those abroad we owe a keen sense of gratitude for our larger conception and a basis for reasoning whereby we are able with our present adjuncts to diagnose and by surgery to give relief and comfort.

At the present time the most frequent operation in all ear, nose and throat surgery is the removal of tonsils and adenoids, and justly so, for there is no question that more pathology is found in these structures than in any other in our field. Not only is pathology found in these structures, but, by its presence in them, infection is transmitted to other structures and causes general systemic trouble or perchance local trouble distant from the original focus. Much has been written on focal infection, and I have no desire to add to the voluminous

literature on the subject. I have had glaring failures and brilliant successes, as many others have had.

An experience of something over 10,000 tonsillectomies that I have done because of clinical findings convinces me not only of the reliability of clinical findings as a basis for tonsillectomy, but has also impressed me with the great responsibility that the surgeon should shoulder in this so commonly performed operation of tonsillectomy, namely, the responsibility not only of leaving a good throat but of removing every vestige of infected tissue.

Impressed with the serious infection in many throats presenting themselves for relief, not a few of which had been previously operated upon by some one or other of the incomplete methods, I have had many tonsils, removed during the past four years, subjected to careful histologic and bacteriologic examination.

It is the purpose of this paper to report a resumé of the findings in this series of cases and to add a word or two relative to impressions gained therefrom.

It might be of interest to state, in a preliminary way, something of the method employed in the pathologic examination. The tonsils, immediately after extirpation, were put into sterile gauze by the surgical nurse and taken to the laboratory, where they were fixed in 10 per cent neutral formol. Adenoids were similarly treated. The faucial tonsils were split longitudinally into two equal parts, the sections being taken from the cut face of one part. The other part was retained for possible further study. The ordinary methods of paraffin infiltration and embedding were employed, and the tissues were stained in hematoxylin and eosin. Usually only one section from each tonsil was examined microscopically, although in certain doubtful cases it was found necessary to section all of both tonsils at intervals of about 2 mm.

The exact number of cases so examined was 2321. Aside from the ordinary pyogenic infection, present to a greater or less extent in all tonsils, the most common pathologic condition found was tuberculosis, involving usually either one or both tonsils. In addition, syphilis was demonstrated in one case where spirochetæ were found in large numbers. There were two cases of rather rare mycotic pharyngitis, which involved

the tonsils, and one case of tumor, namely, perivascular endothelioma, in an elderly person, which had metastasized to lymph glands.

As regards the histopathology of tonsils showing tuberculous infection, they fell for the most part into two main groups, although there were a very few cases of a third group, which might even be considered as a subgroup of one of the main divisions. The first group was characterized by the location of tubercles about a crypt or its ramifications. Sometimes they appeared at the base of the crypt near the capsule, while at other times they were more superficially located, either about the mouth of a crypt or beneath the mucosa of the tonsil itself. In one instance the tubercle was traced by serial sections directly into a crypt, which itself was somewhat cystlike from obstruction. Usually this type of tubercle was epitheloid, sometimes containing giant cells, at other times not. Precisely the same type of lesion was at times present in the adenoids, either in conjunction with the tonsils or exclusively in the former. In five cases tuberculosis was found exclusively in adenoids. Practically never was there any considerable amount of caseation in these tubercles and usually none. At times these tubercles appear to coalesce near the surface of the tonsil, with more or less caseation, resulting in erosion and producing tubercular ulcer. This type of case constituted a subdivision of the socalled cryptic type. The cryptic type of case is usually unilateral.

The other main group of cases is characterized by a rather diffuse dissemination of tubercles throughout the tonsil. They do not respect the germ centers, invasion of which structure has not been observed with the cryptic type. Giant cells are more common, and caseation and secondary infections are very frequent. In this group usually both the tonsils and sometimes the adenoids are involved in the manner stated.

It is highly probable that the latter group of cases is secondarily infected, and is but a manifestation of an active tuberculosis elsewhere in the body. On the other hand, it is more probable that the cryptic group represents a primary infection of the tonsil, either by inspiration or ingestion, or perhaps an infection from open pulmonary lesion. Theoretically it is impossible to rule out the presence of tuberculosis elsewhere

in the body, nevertheless, the relation of these tubercles to the crypts themselves, coupled with the usual absence of clinical signs and with the good follow up histories that most of these patients present, makes it highly probable that such cases have not been infected from an open lesion but rather from inspiration or ingestion. It is very probable that some cases of disseminated tuberculosis may have their crypts infected from an open pulmonary lesion resulting in a mixture of the two types of cases. The clinical significance, however, of such cases would be the same as of the disseminated type.

That such pathologic grouping of the cases may have clinical significance has also been suggested by others, in which event such a pathologic report should be of value both to the clinician and to the patient. The cryptic type represents a relative benign infection, as shown by its relative freedom from caseation and secondary infection as well as by its superficial character. With proper watching such cases promised to have no further involvement. With the other group, to be sure, the clinical significance is different. If evidence of active tuberculosis has not been discovered before, such evidence should afford most helpful information for both clinician and the patient. The significance would, of course, be that of any case of active tuberculosis.

Among the rarer conditions of interest were two cases of mycotic pharyngitis involving the tonsils. Clinically, the appearance of the throat was characterized by discrete, raised, yellowish nodules, which were so adherent as to make removal impossible except by the use of a sharp instrument. Section of these nodules disclosed the fact that a hyperkeratosis was present. This process began at the lower layer of the mucosa, which fact explained the tenacity with which the nodules adhered to it. They presented a lamellated arrangement, and cellular elements were few or absent. Stained by Gram-Weigert, threadlike bacteria were seen between the lamellæ, which corresponded well with direct examination of the exudate and macerated nodules. Long, rather thick threadlike organisms were present in addition to many spirochetes and fusiform bacilli. Cultures were not definitive.

The single case in which syphilis could be definitely proved was one in which the tonsils were enormously enlarged and in

which the condition had not been suspected. The cervical lymphatics were quite palpable, but the patient gave persistently a negative history. Histologically, there were present, particularly in the deeper parts of both tonsils, areas of epitheloid tissue, which were vascular, in contrast to that ordinarily seen in tuberculosis. No giant cells were observed. The Levaditi stain disclosed large numbers of typical spirocheta pallida in these areas and in the lymph vessels, as well as around the blood vessels.

The single case in which a tumor was found occurred in an elderly woman with enlarged lymph nodes. Expiration of both tonsils and nodes disclosed the very rare tumor known as a perivascular endothelioma. Its structure was characterized by the presence of dilated lymph channels lined by polyhedral cells with lymphocytes surrounding the small arterioles, as is the case with a true perithelioma.

Tuberculosis was found in 39 of the 2,321 examinations (1.7 per cent), which is usually lower than in groups studied by others, as follows:

| Author | | T. B. | Per Cent |
|---|---|---|---|
| Albrects | 1,060 | 3 | 0.3 |
| Barnes | 250 | 3 | 2.0 |
| Crowe, Watkins & Rothholz | 1,000 | 46 | 4.6 |
| Judd | 1,000 | 23 | 2.3 |
| Lewin | 200 | 9 | 4.5 |
| Robertson | 232 | 19 (?) | 8.0 |
| Sewall | 772 | 30 | 3.9 |
| Willis | 108 | 9 | 8.0 |
| Weller[1] | 8,697 | 204 | 2.35 |

Virchow, in 1864, stated that tuberculosis of the tonsils had not been observed. Cornil, in 1875, is reported to have found ulcerative lesions of the pharynx and tonsils in an otherwise healthy individual. Deplous, in 1878, found tuberculous ulceration of the tonsils in a child with marked pulmonary tuberculosis. Orth experimentally produced tonsil tuberculosis in dogs by feeding them tuberculous tissue. In 1887, Orth reported well authenticated cases of pulmonary tuberculosis in tonsils of children dying from diphtheria, without evidence of tuberculosis elsewhere in the body.

Tuberculosis of the tonsils has been carefully studied and reported in its many phases by Pluder and Fischer,[2] McBride and Turner,[3] Brieger,[4] Piffl,[5] Rethi,[6] and Hynitzsche.[7] Tonsil tuberculosis occurring during childhood has been investigated by Latham,[8] Friedmann,[19] Ito,[9] Kingsford[10] and Mitchell.[11]

The tonsil as a point of entrance of ingestion; the nature of the infecting organism, whether bovine or human; its relationship to lymphoid hyperplasia and relationship to cervical gland and pulmonary tuberculosis has been dealt with in articles by Gottstein,[12] Grober,[13] Bandelier,[14] Carmichael,[15] Sewall,[16] Mitchell[17] and Crowe, Watkins and Rothholz.[18]

In view of the known effects of tuberculosis or other serious infections of the tonsil, and the incidence of such infection as developed by this and similar studies, the conviction is forced upon one that the question of whether or not to operate in a given case should merit a careful and systematic study of every case presented for examination. In my own experience the method of arriving at such a decision has been a careful noting and recording of the following:

1. History.
2. Clinical examination.

In obtaining the history I have endeavored to obtain the salient facts by the following series of questions:

1. Age.—This seems at first to be a trifling consideration, but there is much diversity of opinion at the present time regarding the function of tonsils. The best advice which I can garner from the literature is that if the tonsils perform any function after birth it has been accomplished before the age of four years, and thereafter the tonsils cease to be a factor in the human economy and become a rudimentary structure. Hypertrophy of the tonsils, during the period up to four years of age, justifies their removal only when they produce a decided mechanical obstruction. At four or five years of age hypertrophied and cryptic tonsils may justify their removal on the grounds of preventive or expectant surgery. Later in life, the more advanced the age the less I advise removal of hypertrophied tonsils, in the absence of serious infection.

2. Difficulty with nasal breathing.—It must be remembered that inabilty to breathe through the nose, due to obstruction

caused by tonsils and adenoids, is rarely encountered in older children at the present time. These cases are not allowed to continue as such, in the light of present informtaion and efficient school examination, welfare work and community measures for their prevention. Adults referred for tonsillectomy because of "difficulty with nasal breathing" may be more in need of nasal surgery than tonsillectomy.

3. Snuffles in childhood and evidence of sinus infection.
4. General health.
5. Frequency of colds and bronchitis.
6. Ear complications.
7. Exanthemata and complications therefrom.
8. Attacks of tonsillitis and their sequelæ.
9. Evidence of cervical adenitis.
10. Appetite and disposition.
11. Possibiilty of hemophiliac.
12. Time that has elapsed since any acute infection and cultures of inflammatory exudate.

Careful consideration of the above points gives one a pretty definite record of any case, and upon the history and information thus gained a pretty definite impression is obtained regarding the pathology to be found.

I cannot emphasize too strongly the cooperation of the internist. In all of my cases a complete physical examination is made just a few days before the operation is performed. To be sure, we operate on cases which show heart and kidney lesions, as in many cases the tonsils seem to be the causative lesion, but not infrequently some acute condition is found of a bronchial nature which renders it advisable to defer the risk of anesthesia for a few days.

In examinations of nose and throat cases attention is first directed to the cervical glands. The presence of enlargements, acute or chronic, with a definite knowledge of anatomic relations, together with the lymphatic drainage, will help to localize the pathology if present. Attention is next directed to the general condition of the mouth, gums, teeth and tongue. A furred, tooth marked tongue is usually an indication of the presence of mouth infection. Should there be present marked irregularities of the edges of the front teeth, a Wassermann examination should be made. The external exam-

ination of glands of the neck and mouth not only gives valuable information, but also tends to inspire the patient's confidence in the examination and dispels much of the fear with which the heart of a youngster, particularly, is filled when anticipating such examinations.

The condition of the pillars, uvula, soft palate and posterior pharyngeal wall is next noted. The information gained from this portion of the examination is of inestimable value. In most cases where infection is present there will be a thickening of the pillars with redness and chronic inflammation of the mucosa covering them. The uvula is often markedly increased in diameter and length, with thickening and redness of the entire soft palate. The lymphoid tissue in the posterior pharyngeal wall is swollen and red, and islets of such tissue are clearly defined. Examination of the tonsil itself consists in noting the size, consistency and cryptic contents. Simple hypertrophy causing mechanical obstruction hås already been alluded to. Previous inflammatory conditions will be evidenced by the replacement of normal tonsil tissue by cicatricial tissue and contractions. The cryptic contents should be carefully expressed by pressure against the anterior pillar and the contents carefully noted. If there is any question regarding the substance thus expressed it is advisable to have cultures and microscopic examinations made. It must be remembered that the cheesy masses which are found in 95 per cent of the cases are not pus and must be differentiated from it. If pus is present it is most frequently noted coming from the upper crypts of the tonsil. This the most submerged portion and where the crypts are more nearly closed as a result of their being covered by surrounding structures. One examination is not always sufficient to demonstrate the presence of pus.

Infection in adenoids is not frequently mentioned in literature, but is present more often than generally believed, and is manifested by marked redness and inflammation of the adenoid tissue and structures in close proximity thereto. Profuse nasal secretion often results.

Presence of adenoids results in a variety of symptoms, which must be considered in the same manner as conditions resulting from tonsils. Adenoids must be considered from the standpoint of the obstruction caused by them, the infection

present and their relation to pathology found in surrounding structures. It must be remembered, however, that adenoids are found normally in every individual at birth and have a tendency to atrophy after the fourth year, therefore, their presence previous to the fourth year is not necessarily an indication for their removal unless some good definite reason can be found for so doing.

The benefits derived from the removal of tonsils and adenoids, whether on account of obstruction or a focus of infection, are many, and its effect on clearing up acute and chronic catarrhal infections of the eustachian tube and middle ear is well understood. Where there is found sufficient reason for the removal of tonsils and adenoids, but at the same time there is found hypertrophy of the turbinates or a slight sinus infection, I have made it a rule to remove the tonsils and adenoids and follow these cases from four to six weeks with cleansing treatments and attention to drainage of sinuses and have been gratified to know how many of these cases clear up without surgical nasal interference.

The technic of operation for the removal of tonsils and adenoids must be decided by the personal equation. The operator must find and utilize the method whereby he can remove the structure completely without mutilation of the surrounding tissue and which gives the most perfect postoperative result. To discuss the different methods would be a long story. I have used nearly every method and have finally decided on the scissor dissection as the method par excellence in my hands. By this method the cleanest and most definite separation of the tonsil from the surrounding structures is made and the postoperative results are most satisfactory.

## CONCLUSION.

The histologic and bacteriologic examination of 2,321 consecutive specimens of tonsils and adenoids removed during the past four years has developed certain facts and impressions as follows:

1. Tuberculosis of tonsils was found in 39 cases, or 1.7 per cent of all cases examined. This is somewhat lower than the average per cent of other available studies.
2. Syphilis is comparatively rare.

3. Malignancy was found in only one case.

4. The great percentage of tonsils demanding removal for clinical reasons, either because of infection or on account of obstruction caused by their hypertrophy, show either simple hyperplasia or infection with pyogenic organisms.

5. Tuberculosis, syphilis, malignancy, pyogenic and other infections found in tonsils removed impresses the writer with the importance of

(a) Thorough clinical study of the individual case before operation.

(b) Thorough operation to accomplish complete removal, with due respect for the surrounding structures.

(c) Careful study of tissue removed.

(d) Thorough follow up work.

I gratefully acknowledge my indebtedness to Dr. R. R. Mellon for his cooperation in the pathologic work.

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## LXIII.

# RADICAL MASTOIDECTOMY; COMPLETE HEALING. METHOD OF CONTROLLING PERSISTENT VERTIGO.

By J. L. Maybaum, M. D.,

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Hugh B., aged 26 years, was admitted to Manhattan Eye, Ear and Throat Hospital, service of Dr. Berens, June 10, 1919, with a history of distressing vertigo and falling, of six months' duration, discharging ears and impaired hearing.

The patient had the usual diseases of childhood. Double middle ear suppuration dated back thirteen years following diphtheria. From then until the onset of vertigo six months ago, except for aural discharge and impaired hearing, there was no camplaint referable to the ears. The dizzy attacks at the outset occurred at infrequent intervals until six months before entering the hospital. The attacks then became of greater severity, occurring daily and being accompanied by falling, particularly to the right.

Aural examination disclosed a moderate foul smelling discharge from each middle ear, with perforation and granulations in posterior superior quadrant. Hearing in the right ear markedly diminished, whisper not heard; C (128) and $C_4$ tuning forks not heard. C—A., 4 sec.; B., 15 sec. Weber to the right. Mixed form of deafness—advanced middle ear deafness with degenerative cochlear changes.

Hearing in the left ear fairly good; whisper, 8 feet; Rinne, negative; high tones as well as the low and middle tones slightly reduced.

There was a spontaneous nystagmus to the left; no spontaneous past pointing. Fistula test negative.

Turning to right—horizontal nystagmus to the left, 20 sec. Turning to left—nystagmus to right, 22 sec.

Caloric stimulation of the right and left horizontal and vertical canals gave normal responses for nystagmus, past pointing and vertigo. Fistula test negative.

I did a radical mastoid operation on the right side. Found a sclerosed mastoid and a cholesteatomatous mass in antrum and aditus and granulation in middle ear removed. The patient left the hospital on the tenth day decidedly better, but still complained of occasional milder attacks of vertigo.

The spontaneous nystagmus disappeared at the end of two weeks, the hearing in the right ear remained as before the operation. The patient came to the clinic for two or three dressings; did not appear again for six weeks. He then returned with the story of having been unable to attend the clinic because of severe attacks of acute articular rheumatism. During this period the ear had been dressed in a more or less perfunctory manner by his family physician. The vertiginous symptoms had recurred with almost as marked an intensity as previous to the operation.

Examination of the operated ear now disclosed an almost complete atresia of the meatus and the canal filled with partly organized granulation and connective tissue. There was a fistula through which a thin purulent foul smelling discharge was escaping.

Hearing very much impaired; could hear only loudly spoken voice with noise apparatus in left ear. Tuning forks not heard. Galton whistle heard.

Caloric test (right ear) ineffective because of the partial atresia of the canal. Caloric (left ear) normal response. A spontaneous nystagmus to the left present. Turning to the right ten times in twenty seconds resulted in horizontal nystagmus to left of 18 seconds' duration. Turning to the left nystagmus to the right 20 seconds' duration.

Patient was readmitted to the hospital. I reopened the postauricular wound. Curetted granulation in meatomastoid cavity and did a plastic operation.

Patient left the hospital in a few days; meatomastoid cavity completely epidermatized in eight weeks. Until the end of May, 1922, the patient continued to complain of vertigo; at times this became so distressing that he had to discontinue his usual activities. Enforced rest gave only temporary relief. The symptoms described by the patient were definitely those of a systematized vertigo. Occasionally these attacks were

accompanied by nausea, but never vomiting. The spontaneous nystagmus ceased three weeks after the second operation and was not again observed.

Functional examination October 11, 1922:

Whisper

| Right | Left |
|---|---|
| 0 | 15 feet |

lateralizes to the right

| Right | Left |
|---|---|
| C-A :8 sec. B :25 sec. | C-A :15 B :25 |
| Other forks not heard. | C-6 sec. |
| Voice heard with noise apparatus in left ear. | Caloric not performed (dry perforation). |
| Caloric positive in 35 sec. | |

Turning to right, horizontal nystagmus to left 22 seconds.

Turning to left, horizontal nystagmus to right 18 seconds.

Pastpointing and falling normal.

Normal reactions on testing vertical canals.

Every possible factor causing these attacks of vertigo were considered in order to give relief to the patient.

1. A continuance of irritation from the paralabyrinthine inflammation in the operated ear was constantly kept in mind With a complete healing of the radical mastoid cavity, the disappearance of spontaneous nystagmus and normal caloric response, this factor had to be eliminated.

2. A paralabyrinthine inflammation in the nonoperated (left) ear was for a time considered. Because of the marked diminution of hearing in the operated (right) ear, I was loath to seriously consider this factor from the standpoint of operative interference. The presence of comparatively good hearing in this ear (whisper 8 feet), together with absence of spontaneous nystagmus, pointed against the left ear as a causative factor. Under treatment this ear has further improved; there had been little, at times no discharge during the past year.

3. Both vestibular nerves and their intracranial pathways gave normal responses for nystagmus, past pointing and vertigo.

4. Cerebrospinal lues has been ruled out, Wasserman being negative.

5. Other extraneous factors, as local infection, gastrointestinal disease, errors of refraction, were carefully considered and eliminated as causes.

6. Vasomotor disturbance of the labyrinth. After unsuccessfully running through the gamut of the various remedies advised for the relief of these patients, such as small doses of quinin, large doses of bromides, pilocarpin, etc., in an empirical way, I began the use of solution adrenalin 1-1000, ten drops T. I. D., by mouth, as suggested by Vernet in his article, "The Treatment of Vertigo by Means of Adrenalin," appearing in the April, 1922, issue of the Laryngoscope. In a few days the patient obtained complete relief from his annoying symptoms. Withdrawal of the adrenalin for a few days after its administration for ten days was followed by a return of vertigo, but to a lesser degree. Adrenalin medication has been discontinued with no recurrence of vertigo. His general condition has steadily improved during the past few months.

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LXIV.

# SOME OF THE ASPECTS OF APHONIA, PSYCHIC AND OTHERWISE.

By James A. Babbett, M. D.,

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The subject of aphonia presents a rare composite of analytic research and routine laryngology. The problem of voice recovery may require the most painstaking study of motor, sensory and ganglionic innervation, investigation of the whole field of metabolism complex, with laboratory findings, and yet equally emphasize the simple office corollaries involved in obtaining the patient's cooperation, suggesting confidence, recognizing idiosyncrasy and, not least, holding the proper perspective toward the factors governing his social and economic efficiency. The field of the topic is purposely a broad one. It is the intention here, however, to study the sequence in aphonia, trace the etiologic factors, analyze briefly the physiologic and anatomic alterations involved and stimulate discussion on the prognosis and remedial measures, not in the fixed aphonias of malignancy, advanced luetic and tubercular dyscrasias, but rather in the psychic and functional types, often exasperatingly fixed in character, and in those arrested structural losses where vocal reeducation, automatic or directed, becomes a possibility—perhaps more concisely expressed, on the basis of etiology and sequence, estimate the balance of possibility in restoration.

From the anatomic and physiologic standpoint the mechanism of phonation depends upon the sliding and rotation of the three surfaced arytenoid cartilage to whose anterior or vocal process the cords are attached, and to whose external or muscular process the intrinsic musculature is inserted. To briefly review, the cricothyroid with more or less vertical and horizontal fibers tightens the cords by the raising of its anterior and tilting downward of its posterior half. This muscle practically alone receives its innervation from the external branch of the superior laryngeal nerve. The internal branch

of the superior laryngeal affords sensory supply to internal surfaces of the larynx, including the ventricular space of Morgagni—true and false cords. Its antagonist is the thyro-arytenoid muscle which has two planes, internal and external, which may act independently in the falsetto voice, which might perhaps be considered a threshold to aphonia. The crico-arytenoid posticus and lateralis, by external and internal rotation of the arytenoid respectively abduct (widen) and adduct (narrow) the rimal slit. The posterior arytenoid crossed and horizontal fibers draw the arytenoids together and complete the tightened closure. All of these latter muscles depend upon the recurrent laryngeal for innervation.

Holmes Jackson's careful analysis of the "Reflexes From the Larynx" shows the complexity of neurologic factors involved and emphasizes the corollation of the glossopharyngeal and respiratory center in the medulla. Careful study of this mechanism makes it clear that in the types under discussion complete aphonia can scarcely be separated from partial and intermittent phases, for we are considering types capable of voice restoration. This may include deviation in muscular structure, vocal cord surface and any encroachment upon intralaryngeal space as well as nerve integrity.

Etiologic factors in aphonia, while in general of central, peripheral and toxic origin, as grouped by Lederman, present a marvelous complexity of basis factors. In the central group might be included bulbar lesions, thrombosis, embolism and hemorrhage, brain tumors and intracranial growths at base, multiple and lateral sclerosis, parasyphilis and syphilitic gummata, glossolabiolaryngeal paralysis, tabes dorsalis, lesions involving the vagus and recurrent, every form of cervical pressure and trauma, mediastinal irregularities of varied sort, aneurysms, pericordial and peribronchial effusion, enlarged thymus, diseased thyroid, constriction of postoperative scars and ligatures, pleural thickening in apex, principally right, dilatation of left auricle, diseases of mediastinal glands, carcinoma, sarcoma, lymphosarcoma, syphilis and tuberculosis, involvements of esophagus, cervical adenitis, etc. Dr. Cress has reported from recent Vienna clinic observations additions to this list.

From a practical standpoint, the third and toxic group have a more practical application to the laryngologist in our present emphasis upon focal infection—varied toxins of mineral and vegetable origin, lead, arsenic, potassium iodid, iodoform, alcohol, morphin, crude opium, cocain, belladonna; toxins from the acute infectious diseases—pneumonia, typhoid, diphtheria, puerperal fever, measles, gonorrhea, influenza, acute rheumatic fever, disease of thyroid, metastasis from infection of tonsils, teeth, sinuses, middle ear, broken down cervical glands, with more often perhaps streptococcic infection. Under this heading might be included poisoning from inhaling gases, of which so much postwar history has been quoted by Milligan, Hurst and others.

Tuberculosis must be given an important place, from both its mediastinal influence and its contribution to the asthenia so common in hysterical or functional aphonia. Garland and White reported, in 1920, a series of nine cases of paralysis of the left recurrent laryngeal associated with mitral stenosis, in the Massachusetts General Hospital during the past eight years. They consider the record of only 61 cases in the literature, including theirs, as indicative that many are overlooked. A great variety of special etiologic types have been also recently reported, such as: Eversion of the Ventricle and Saccule, by Moore and Tapia; Relaxation of the Diaphragm, by Stoerck; Amyloid Degeneration, by Bruzzi, Evers, Ackerman and Harbitz. Time prevents further enumeration.

With this basis of fundamental mechanism and complex group of etiologic factors, it is our purpose to discuss three important aspects in aphonia, quoting one or two selected illustrative subjects: (1) The metabolism complex, or syndrome in aphonia; (2) the borderline difficulty in apparent psychic types; and (3) the phonation prognosis after arrested tissue loss.

1. The Metabolism Syndrome.—Gill in a recent address before the section in Philadelphia emphasized the importance of the metabolism factors in arthritic cases which per se should have been relieved by surgery of the teeth, infected tonsils and sinuses—with such a manifold group of etiologic factors as we find active in types of aphonia often not simply central, peripheral or toxic, but varied combination of these, pro-

foundly subject to endocrin and basal metabolism influence, it is reasonable to link functional aphonia to general metabolism. From the simple aphonia of acute laryngitis to the paralysis from recurrent laryngeal bloc, at least circulatory metabolism is impressive. The nature of a nerve impulse is obscure, the chemistry of its root cell not much less so, but trophic influence of nutrition upon both will be admitted. Toxemia, zymolysis, acidosis, endocrin changes must affect this neuromuscular action, and particularly in myopathic types. The carbohydrate balance regulation of the posterior pituitary lobe grow stimulation and sexual activity, control of the anterior, have a reasonable relation to psychic aphonia.

One of the reasons for the inception of this paper was the difficulty experienced in recent years in determining the basis of aphonia in borderline cases, and to stimulate discussion upon the same the following case is cited for illustration:

Case 1.—In July, 1920, Carrie G., referred by Dr. B. Girl of 15, slender, phlegmatic rather than neurotic in tendency. Early history negative except for series of spasms reported at three years of age and suppurative otitis media, right side. Somewhat later, under medical care for reputed cardiac weakness. Voice gradually disappeared nine days prior to visit. Upon examination, larynx apparently normal. August, 1920, tonsils and adenoids removed at Lankenau Hospital. September, 1920, digital avulsion of cords, resulting in modified phonation half of one day. Physical, neurologic, fluoroscopic, laboratory urine and blood examinations all negative. Tonics, thyroid, pituitrin and ovarian extracts and iron injections administered. No result. Wassermann negative. Massage and electricity ineffectual. In December, 1922, a typical reflex cough appeared, apparently due to recurrent laryngeal irritation, and persisted for three or four weeks. During this time the laryngoscopic picture presented a normal appearance. Adduction and tension bilaterally equally failed to produce tone. Cords were apparently approximated, though somewhat weakly, and lower triangular area sometimes remained open. According to Semon-Rosenback law, indications all point to functional aphonia.

The significant premise in this recital is that the voice has failed to return except for this one-half day, and now and

then a very incidental approximate tone, for a period now of almost three years. Within the past month a further somewhat exhaustive study has been made. Larynx still appears somewhat weak. No obvious fault in the laryngeal cavity. Careful X-ray examination by Dr. Pfahler shows sinuses frontal, sphenoid, maxillary clear, slight amount of exudate in ethmoids. A somewhat deflected septum and spur may be related to this, but patient declines operative relief for this. Sella turcica normal. No constriction of the trachea from pharynx downward. No mediastinal tumor or gland. A few calcified lymph nodes in mediastinum about hilus of lungs but no evidence of pulmonary disease. Heart and aorta normal. External examination of chest and heart also normal. Barany tests were taken in intercranial search. Horizontal rotation nystagmus, past pointing and vertigo normal. Douching right ear with cold water—vertical position, no response for five minutes; with head back, slight horizontal nystagmus appeared. Douching left—good response, 1 min. 45 sec. Ocular, falling, Rhomberg and reflexes normal. Recent blood examination and basal metabolism all negative; erythrocyte count, however, 4,000,000.

These reports have been only briefly reviewed to cite a typical case of difficulty in borderline cases. With one exception (of Barany rotary nystagmus after douching in an ear where acoustic labyrinth was unaffected) all symptoms point to functional aphonia, yet one in which the metabolism complex apparently aids in rendering the aphonia static. The serious influence in the patient's life regime is obvious. The matter of its organization would be simple were the basic evidence of some central difficulty or pressure neurosis. The case seems a complex involving perhaps endocrins and peripheral circulation and idiopathic asthenia, and would perhaps be grouped as a case of type reported by Stern, photoasthenia.

The concluding aspect of aphonia to be considered is that of the natural effort of the larynx and its associated structures to get well. The marvelous results in reeducation of the voice after total laryngectomy impress the possibility in this direction. The Early case reported by Cohen and the experiences of varied operators more recently attest this farther. In illustration of this phase the following history may serve:

Case 2.—In March, 1922, was called to see patient, Mr. J., who had developed increasing difficulty in phonation, and even some dyspnea without pulmonary complication following a major abdominal operation. The abdominal operation was reported as malignant, requiring removal of a segment of intestine—in fact, a portion of the malignant area was inoperable. The patient had some indefinite previous laryngeal history. As his legal profession demanded much public speaking, his previous trouble was probably of no organic nature. Certainly there was no specific, malignant or tubercular history locally.

In recovering from the above cited condition, at which time the stomach tube had been used, the patient experienced excessive retching and vomiting, and to control this repeatedly clutched the throat and larynx with finger far back in the pharynx, infectively and traumatically complicating the picture. The patient when I visited him had complete aphonia and rather alarming dyspnea, and his larynx presented a picture of general congestion, edema, mucopurulent secretion and irregularly dislodged slough, entirely filling the ventricular cavities and obscuring all view of cords and arytenoids.

Careful examination by cultures and smears only revealed mixed infection largely staphylococci, and no t. b. bacilli were found. Under local treatment, administered with care on account of edema and dyspnea, the congestion gradually subsided, first right and then left cord came into view and finally the arytenoid area. In the meantime Wassermann test was taken and found negative. Voice gradually was recovered, though left cord showed apparent tendency to abductor paralysis. In breathing, the position was usually miocadaveric, though the article by Fein would deny this term.

On March 28th, eighteen days after the original examination, patient coughed out what appeared to be left arytenoid cartilage, and, two or three days later, the right. X-ray, fluoroscopic and laboratory examination confirmed this diagnosis. Patient was profoundly aphonic at this time, though dyspnea had improved. About three weeks later patient commenced to recover voice, and in six weeks produced fair tone, low pitch for the most part, and variable in force. In June the patient was placed under care of a New York laryngologist,

his larynx capable of moderate tone production, alteration in the arytenoid field, but no malignant evidence. On November 9th, reported somewhat discouraging reversion to whispered or modified phonation, but patient decidedly asthenic from intraabdominal progress in malignancy.

This case is of interest from the fact that he was recovering acceptable voice in spite of the absence of both arytenoids. The literature is replete with instances of recovery of voice after removal of one or even both cords and total laryngectomy, with subsequent voice reeducation. Prof. Chiari reported in 1909 before the society his results after operations for malignance. Time will not permit us to discuss this in further detail.

## CONCLUSIONS.

1. Partial and intermittent types of loss of phonation may properly be considered with aphonia and the latter not considered a fixed aphonia except in a limited group of conditions.
2. The complex changes of metabolism, variation in endocrins, zymolysis, acidosis, toxemia, etc., may profoundly influence functional aphonia.
3. The complex symptomatology of borderline cases, organic vs. functional aphonia, renders diagnosis and prognosis extremely difficult.
4. The reconstructive forces in restoration of phonation after arrested pathology, even with marked tissue loss, are most surprising.

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LXV.

# FOREIGN BODIES IN UPPER AIR AND FOOD PASSAGES—A CLINICAL REPORT.*

By Harrington B. Graham, M. D.,

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What Jackson said in 1915 concerning the armamentarium in bronchoscopic or esophagoscopic work is still pertinent in spite of the advances made in the mechanics in this field. He then said that there was no one set of instruments which could be considered the best for all work or for all men; that it was more the man behind the instruments that was going to save life, rather than the instrument itself. If one has at his disposal the large tubes of Mosher as well as the small ones of Jackson and has practiced with both, he will certainly choose the Mosher tubes for the examination of an esophageal carcinoma in an adult and the Jackson tubes for a foreign body in the bronchus of an infant.

The advantages of a proximally lighted tube for diagnostic purposes are, it seems to me, beyond a doubt but the disadvantage in the dispersion of light from an instrument introduced into this tube during the removal of a foreign body tends to render it far less serviceable than the distally lighted tube. Skill in the handling of any of the instruments can only come with painstaking practice, and the best results will certainly follow through a skill acquired in the use of a variety of instruments. Our ultimate purpose is to save life with the least damage to the tissues involved and with the least strain on the patient; not to satisfy our egotism by a claim that a foreign body has been removed with the smallest tube in the shortest time on record. If by using a small tube the patient is subjected to three seances, whereas had a larger tube been used, it could have been removed in one, it were better to have chosen the larger diameter tube. Each case must be judged

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by itself, and that method or instrument selected which will best fit the circumstances and conform to the skill of the operator.

In analyzing my cases, I have been surprised at the large number of unorthodox methods used, and still out of 43 cases of foreign bodies there has been but one death, a mortality of 2.3 per cent or 5.8 per cent of the bronchoscopies. For instance, in two cases (Nos. 1 and 32) I resorted to a low tracheotomy in order to reach the foreign body; in both cases a prolonged effort had been made to get the article and in each case it was readily extracted through a tracheotomy opening. Again a fluoroscopic bronchoscopy was done twice, once unsuccessfully and once with a most satisfactory result. The successful case (No. 33) had been tried under local anesthesia a year before, but there was such a large quantity of pus present that there was but little chance of locating the article, a shirt stud, by vision. Under ether with the fluoroscope a tube was passed readily to the article and later a forceps which was closed upon the stud and a successful result obtained. The stud had been in the left lower lobe for twenty-three years and granulation tissue had formed over it. No effort was made to remove the granulations, as Jackson had done in a similar case, as the probe passed so easily. At no time was the foreign body seen through the bronchoscope, but the forceps seemed to enter a cavity when passed to it, and there seemed to be little chance of grasping lung tissue.

Unorthodox methods do not necessarily mean poor judgment. They may mean a less skillful technic, but perhaps a more rational safety for the patient. I have seen a little patient go back to bed in thorough shock after a struggle with the overzealous staff of physicians who were attempting to pass a tube without an anesthetic, whereas a little ether would have quieted the nerves of both the physicians and bystanders, to say nothing of the patient. It is one of the triumphs of surgery that this can be done so easily by the skilled endoscopist. It is one of the tragedies that it should be tried by the less skilled in a well intentioned but ill advised effort to save the child or to show his prowess.

There have been foreign bodies which, if dislodged, would close off the only lung tissue which was functioning where it

was extremely desirable to operate without an anesthetic, but these cases are few and far between, and I believe with the bronchoscope in such universal use as at present that it would be far wiser to advise the use of an anesthetic until the requisite skill had been acquired. After the tube has once been introduced we all have found that it takes but little ether to continue the work indefinitely, especially if cocain is used at the distal end of the tube. All of my cases under 6 years of age, save one, two cases of the five between 6 and 20 years of age and two of the 25 above 20 years of age were etherized. All of the others were cocainized save a baby of 8 months, on whom an esophagoscope was passed. For diagnostic or treatment purposes strictures, carcinoma, thyroid, asthma, a number of cases have been examined without an anesthetic but with indifferent success.

In the diagnoses of these cases some interesting facts were brought out. In five cases (2, 21, 27, 32, 38; metal whistle, gold crown, primer, pine nut, blanched almond) a whistling could be heard, in three near the patient and in two across the room; in each case it had been observed by the family before seeking the doctor. This whistle immediately disappeared on removal of the foreign body. It has been the solid bodies which nearly occluded the main bronchus which apparently produced this whistling sound.

In Case 3 the patient insisted for years that he had a tack in his lung but was laughed at by his doctors. It remained in twenty years, causing hemoptosis, night sweats, pus expectoration and fever.

Case 33 was accepted in the army after an extensive lung examination, including a radiologic examination on account of the claim of the patient. After entering the army further X-ray pictures were taken with a negative result. After discharge from the army, at which time he says he was declared to have had normal lungs, he developed a lung abscess and fell into my hands. A radiograph showed clearly the metal shirt stud in the left lower lobe bronchus of about the fourth degree.

In Case 32 the radiograph showed an uneven edge at the top of the primer which should have been interpreted as a

closure of the top. The error in not so interpreting it cost the child a number of seances. Too much stress cannot be laid upon a prolonged and careful study of every element connected with the diagnosis of the character, size, shape and location of the intruder before any attempt is made whatever at removal, for at times the simplest element may be the secret that effects the solution of the problem. A hurried diagnosis and an eagerness to get at the case has been the only cause, I believe, of many a failure. The mere presence of a foreign body is seldom a tragedy, but a failure to get it at the first sitting may be.

A diagnostic symptom which has been held of value in these cases is a sudden spasmodic cough in a child which has been previously well, but this sign, pronounced in its suddenness, was present in a child of two years in whom no foreign body was found and who passed through a typical attack of lobar pneumonia, so that one cannot rely entirely on such a history.

Special points of interest noted in the treatment of this series of cases are:

Case 17.—A gold clasp pin lying open in the left bronchus of a nine-months-old child was so manipulated as to bring the point of the pin into the tube; the body of the pin was then grasped and pulled through the tube. For two days there was a temperature of 101 to 103 degrees, and considerable difficulty in breathing, relieving by a steam tent. Subsequently the child made an uneventful recovery.

Case 18.—A case of an open safety pin in the upper third of the esophagus of an eight-months-old child. Again the point of the pin was brought into the lumen of the tube, the body of the pin grasped and the pin partially closed and the tube and pin brought out together.

Case 23.—The open safety pin was at the lower end of the esophagus in an eight-months-old child. I had no pin closer which would pass through the tube, and as the surroundings were unfavorable I judged there would be more trauma in an attempt to extract perorally than there would by an incision through the abdominal wall. I therefore passed the esophagoscope after an incision had been made and pushed the pin down onto the finger of the surgeon. The whole operation took twenty minutes and the child made an uneventful recovery.

Case 22 was that of a bullet in the antrum. The surgeon, while operating under ether with a fluoroscope, lost the lead, which dropped into the right bronchus. I was called to the hospital and introduced the tube while the patient was still under the ether. It was a strange and fortunate coincidence that where a fluoroscope was so necessary it was being used for another purpose.

Case 24 was a series of tragedies. The patient, an adult, had swallowed a chicken bone, which had lodged in the upper portion of the esophagus, and after three days produced an abscess in the neck. The bone was removed under local anesthesia, but in so doing the porcelain tip of an incisor tooth was broken off and slipped outside of the tube into the abscess cavity. Three unsuccessful efforts were made to reach it perorally, during one of which the tip of one of the forceps broke off, lodging in the esophagus and necessitating its removal. An effort was now made to reach the porcelain through an incision in the neck. At no time were we able to feel the article, but the manipulation evidently forced it into the esophagus, as an X-ray subsequent to our operation showed an absence of the porcelain. An ill wind seemed to be following this patient, as, while I was working on her, her mother aspirated a small fruit pit and was about to submit to a bronchoscopy when, after three days, she coughed it up.

Case 25 was a wishbone which had been pushed into the larynx by digital manipulation, lodging in the sinuses of Morgagni in such a way as to be nearly invisible to the mirror. It was extracted by the indirect method, causing a sensation of astonishment to the operator as it gradually unfolded itself. Part of the bone was broken off after extraction, so it does not show in toto in the illustration.

Case 27.—A gold crown was found in the left bronchus of an adult with the open portion up so that it was easily grasped. It would not pass through the tube, however, and caught at the glottis. An effort was made to manipulate it through the glottis, during which it slipped off the forceps, dropping fortunately into the esophagus, and was promptly swallowed.

Case 32.—A child of 4 years, with a 22 primer in the right bronchus, had four bronchoscopies (three by myself) without

the operators seeing the foreign body. The only explanation I can give is that the primer was lying close to the lower wall and fitted so snugly that it appeared to be the carena itself. Through a low tracheotomy and a large tube there was no difficulty about locating and removing it.

Case 39 had been shot in the face fifteen years before and had not known that he carried the lead about with him. He came to me complaining of a soreness in the posterior pharyngeal wall, and an X-ray revealed three pieces of lead, one of which was producing an ulceration. This one was easily removed but the other two were not located even under ether.

Case 40, while driving a machine on the highway, suddenly was cognizant of a stinging sensation in the throat and an inability to talk. On examination it was found there was blood on the skin over the larynx, and he was taken to the hospital. An X-ray revealed a 22 bullet imbedded in the larynx wall, evidently a chance spent shot. A surgeon made an unsuccessful attempt to remove it through an external incision when I was permitted to see the case. To date the body remains in situ. An unsuccessful bronchoscopy was done by another surgeon in an effort to get the article.

Case 41.—A child of 15 months had inspired a tack, which had lodged in the right bronchus, and the X-ray showed it at the level of the diaphragm posteriorly. A bronchoscopy did not reveal the tack, and the child developed a pneumonia, from which she died five days later.

Case 42 came to the hospital eight days after having swallowed a furniture tack. One attempt had been made to get it, but unsuccessfully. X-ray revealed the tack in the left lower lobe bronchus. There was a temperature of 104, with remission to nearly normal each day, and signs of pneumonia, with leucocytes of 26,000, so that there was a delay of five days before a bronchoscopy was attempted. As the temperature did not go down a successful attempt was made to extract the tack, the temperature immediately dropping to normal and remaining there.

The first fourteen of these cases were reported in the California State Journal of Medicine, September, 1916.

| | Age | Foreign Body | Time of Sojourn | Location | Tube | Anesthetic | Forceps | Result | Time | Route | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 15. Dr. E. | 30 yrs. | Fish bone | 2 hrs. | upper ⅓ esophagus | 10 | cocain | plain | removal | 5 min. | peroral | |
| 16. Griffin | 30 yrs. | chicken bone | 2 hrs. | upper ⅓ esophagus | 10 | cocain | plain | removal | 5 min. | peroral | |
| 17. Chollete | 9 mo. | gold beauty pin | 4 da. | l. lower lobe bronchus | 7 | chloroform | plain | removal | 20 min. | peroral | Difficulty in breathing following removal. |
| 18. Zeller | 8 mo. | open safety pin | 24 hrs. | upper ⅓ esophagus | 8.5 | ether | plain | removal | 20 min. | peroral | Tube passed by pin once. |
| 19. C. D. | 12 yrs. | rubber pencil tip | 7 da. | l. l. lobe bronchus | 10 | ether | plain | removal | 10 min. | peroral | Attempt made by another surgeon. |
| 20. S. C. | 8 mo. | open safety pin | 2 da. | esophagus | 7 | none | | not seen passed into stomach | | | Baby passed pin in stool. |
| 21. Kalsbeck | 1 yr. | blanched almond | 14 hrs. | trachea at bifurcation | 7 | ether | plain | removed in pieces | 8 min. | peroral | Attempt made by another surgeon. |
| 22. D. C. | 30 yrs. | lead bullet | 1 hr. | right lower lobe bronchus | 10 | ether | plain | removal | 10 min. | peroral | Accident occurred while under ether. |
| 23. E. C. | 3 yrs. | open safety pin | 32 hrs. | lower ⅓ esophagus | 8.5 | ether | plain | removal | 20 min. | gastro-enterostomy | Pin pushed into stomach. |
| 24. Miss B. | 30 yrs. | forceps chicken bone tooth | 5 min. 5 da. ? | upper ⅓ esophagus esophagus | 10 | cocain | plain | removal | | | Three foreign bodies in one case. |
| 25. Eyre | 30 yrs. | chicken bone | 22 hrs. | larynx | | cocain | | removal | 10 min. | peroral | Removed by indirect method. |
| 26. Mrs. F. Smith | 25 yrs. | chicken bone | 23 hrs. | esophagus | 10 | cocain | plain | removal | 5 min. | peroral | Removed by the cocainizing brush. |
| 27. Moloney | 40 yrs. | gold crown | 2 hrs. | l. l. lobe bronchus | 10 | cocain | plain | removal | 10 min. | peroral | Crown dropped into esophagus. |
| 28. Edwaerds | 12 yrs. | watermelon seed | 3 da. | l. lower bronchus | 8.5 | cocain | plain | removal | 10 min. | peroral | |

| | Age | Foreign Body | Time of Sojourn | Location | Tube | Anesthetic | Forceps | Result | Time | Route | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 29. V. C. 1 | 25 yrs. | pin | 1 da. | l. l. lobe bronchus | 10 | cocain | plain | removal | 5 min. | peroral | |
| 30. V. C. 2 | 20 yrs. | chicken bone | 2 hrs. | middle 1/3 esophagus | 12 | cocain | plain | removal | 5 min. | peroral | |
| 31. V. C. 3 | 25 yrs. | meat | 1 da. | esophagus | 12 | cocain | plain | removal | 5 min. | peroral | |
| 32. Riley | 4 yrs. | primer | 4 wks. | R. L. bronchus | 7.5 | ether | plain | removal | 4 attempts 1 hr. each | tracheotomy | One attempt by another surgeon. |
| 33. Archibald | 24 yrs. | shirt stud | 23 yrs. | l. l. lobe bronchus | 10 | ether | double curette | removal | 2 attempts | | Under fluoroscope, former attempt by self. |
| 34. Graham | 3 yrs. | 25c | 3 hrs. | esophagus | 8.5 | ether | plain | removal | 10 min. | peroral | |
| 35. Lamb | 50 yrs. | chicken bone | 12 hrs. | esophagus | 10 | cocain | plain | removal | 5 min. | peroral | |
| 36. F. P., 1 | 20 yrs. | fish plate | 2 hrs. | esophagus | 12 | cocain | plain | removal | 5 min. | peroral | |
| 37. F. P., 2 | 30 yrs. | fish plate | 12 hrs. | esophagus | 12 | cocain | plain | removal | 5 min. | peroral | |
| 38. Cechini | 8 yrs. | pine nut | 4 da. | right bronchus | 8.5 | cocain | plain | removal | 10 min. | peroral | Wheeze heard with mouth open. |
| 39. C. K. | 38 yrs. | lead bullet 3 pieces | 15 yrs. | post pharyngeal wall | | ether cocain | | 2 removals | | peroral | The third could not be found. |
| 40. Englebret | 30 yrs. | spent bullet | 2 da. | larynx wall | | ether | | not found | | | Patient seen in consultation. No attempt by self. |
| 41. J. Miller | 15 mo. | tack | 5 da. | r. l. lobe bronchus level diaphragm | 7 | ether | | died 5 days pneumonia | 1 hr. | | Tack not seen. |
| 42. Rule | 7 yrs. | tack | 13 da. | r. l. lobe bronchus | 8.5 | ether | plain | removal | 15 min. | peroral | Fever of 104 with pneumonia. Fever dropped with removal. |
| 43. Murphy | 40 yrs. | tin bottle cap | | upper esophagus | | cocain | double curette | removal | 1 min. | | Indirect method. |

## LXVI.

# THE COCHLEA-PALPEBRAL REFLEX IN INFANTS.*

By Theodor Demetriades, M. D.,

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The question as to whether or not the newborn hear is dependent upon observing the reaction which follows a sound irritation on the ear. By different investigators this question has, however, been answered in many different ways, due to the fact that different methods of investigation were used in reaching these conclusions

Among the first to investigate this subject must be mentioned Kussmaul, who in a short contribution held "that of all the senses, hearing was the longest to remain dormant." Genzer and Lange held that during the first and second year of life the reaction to acute irritation was already present—in fact, the movements of the whole body but not the cochlea-palpebral reflex. In like manner as both of these authors, Moldenhauer held to the same views. On the other hand, Poli holds that a ten-day old child will respond to a noise; however, he does not specify the distance for this form of reaction. Sachs gives the appearance of the hearing in a child at 7, Gomperz at 1 year old. Kohlreuter could obtain no reaction in the first 24 hours after birth by the use of tuning forks ($C_1$, $C_2$, $C_3$), but could obtain a definite reaction from the Galton whistle. In like manner, Zimmermann arrived at the same conclusions.

Prever holds to the opinion that all newborn are deaf, without exception, both to weak and high tones as well as to noises,

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Translated by Dunbar Roy, A. B., M. D., Atlanta.

*From the Ear Department of the Policlinic in Vienna. Service of Prof. G. Alexander.

The translation of this article was undertaken because the subject is not mentioned in our textbooks on Otology and Ophthalmology. It has seemed of so much importance to me that I feel as if the article should be placed in readable form for English-speaking Otologists. (D. R.)

and that the first reaction to sound irritation appears on the fourth day.

Kutvirt has been one of the most thorough investigators of the hearing changes in the newborn. He proved that while testing the hearing of the newborn (using $C_2$ and $C_3$ tuning forks) he was able both to arouse the infants from sleep or to produce crying or to call forth some general muscular reaction. The aural palpebral reflex was only incidentally investigated by this author, without attaching any importance to the same.

On the other hand, an objection must be raised to these tests by Kutvirt, because of the fact that on account of the various degrees of sleep from which these infants were aroused, the hearing irritation would not work the same in all cases. From these experiments Kutvirt came to the conclusion that the newborn does show reaction to the hearing stimulations at various times afetr birth and, in fact, he observed them one-half hour after birth of the child.

The varying intervals of nonresponse to hearing stimulation, according to Kutvirt, stands in close relationship to the length of the childbirth, and on the ground of his statistics he holds that the longer the interval between the first pain and the birth of the child, the later will the hearing reflex appear. From a survey of the above literature it is evident that up to this time only the general body reactions had been considered as indicating the presence of hearing in the newborn. Now, however, the recent investigations have hinged on the importance of the auropalpebral reflexes as a diagnostic sign for determining the presence of hearing. The question therefore is the relationship of this reflex to the newborn.

Encouraged by Prof. Aexander and having had access to the rich material of the II University Woman's Clinic through the kindness of Prof. Weibel (to whom I hereby acknowledge my thanks), I have tested out the possibility of hearing in 105 newborn in order to determine how long after birth is the appearance of the cochlea-palpebral reflex. In reference to the experimental tests the following is to be noted:

The investigations by Cemach, Wotzilka and Brunner show that positive results from the cochlea-palpebral reflex are

present only with persons who hear, otherwise it would be impossible to draw fixed conclusions.

From a negative result of the reflex, as Cemach has always held, we should be very cautions in drawing a conclusion because it can be absent even in those with normal hearing (8 per cent of cases).

Proceeding now to my own studies as to the results obtained in these investigations, we come to the following conclusion:

If the cochlea-palpebral reflex is produced in a child then it is evident that the child possesses some hearing, but this means that it indicates nothing as to the future degree of hearing in the examined person.

The cochlea-palpebral reflex was produced by striking the tuning fork $C_4$ with a small uncovered percussion hammer against the metal prongs. Just opposite to the method pursued by Kutvirt I tried to get the children for examination while they were fully awake. This was accomplished by the nurse holding the child on her arm and playing with it or the child was awakened out of its sleep by my passing a small ear speculum into the external canal. The tuning fork was struck four or five times close to the ear of one side at intervals of about 15 seconds. The appearance and the intensity of the reflex seemed to be dependent upon the age, the weight and the size of the infant.

The type of reaction was not the same by all children. Some reacted with distinct opening and closure of the eyelids while others moved the whole body as if frightened. Most of the children began to cry following the striking of the tuning forks. In none of the 105 newborn who were examined did the reflex appear immediately after the cutting of the cord (10 cases); the reflex appeared most frequently between the second and third hours after birth (Tables I and II), but usually from the fourth to the sixth hour after birth.

This statement is only in reference to children whose birth was normal and whose parents were healthy. At the latest the reflex appeared during the first 48 hours after birth.

If within this time the reflex could not be produced, usually it was not possible to make it appear even within the next eight to ten days (two normally born children, healthy par-

ents). After this time I could not keep the children under observation because they left the clinic with their mothers.

TIME OF THE APPEARANCE OF THE COCHLEA-PALPEBRAL REFLEX AFTER BIRTH.

TABLE I.

| Number of hours after birth in which the reflex appeared for the first time. | Number of children showing reaction. |
|---|---|
| 2 Hours | 1 |
| 3 Hours | 2 |
| 4 Hours | 15 |
| 5 Hours | 24 |
| 6 Hours | 26 |
| 7 Hours | 2 |
| 8 Hours | 3 |
| 9 Hours | 2 |
| 10 Hours | 1 |
| 11 Hours | 1 |
| 13 Hours | 1 |
| 15 Hours | 1 |
| 16 Hours | 1 |
| 19 Hours | 1 |
| 22 Hours | 2 |

TABLE II.

| Number of days after birth in which the reflex first appeared. | Number of children showing reaction. |
|---|---|
| 1 Day | 3 |
| 6 Days | 1 |
| 7 Days | 3 |
| 8 Days | 3 |
| 9 Days | 3 |
| 11 Days | 2 |
| 12 Days | 1 |
| 13 Days | 1 |
| 16 Days | 1 |
| 19 Days | 1 |

From the investigations of the previously mentioned authors, as well as from my own results, it must certainly be concluded that the cochlea-palpebral reflex is absent during the first few hours after birth.

For an explanation of these results various authors have attributed different causes.

Most of the investigators attribute as the cause for the absence of hearing, to the filling up of the auditory canal with the vernix caseosa. Gomperz and V. Urbantschitsch think that it is due to the narrowness of the canal in the newborn.

According to Vierordt, the late appearance of the hearing in infants is due to the horizontal position of the drum membrane; according to Kutvirt, to the poor elasticity of the membrana tympani, as well as to the edematous material in the middle ear which is known to exist in infants. It is evident, therefore, that these sound conducting impediments are the cause for the late appearance of the hearing function.

If we accept the correctness of these explanations then we must draw the conclusion that the hearing of the newborn will first be evident when these various impediments in the sound conducting apparatus have been removed.

Such a thing is improbable because my previous investigations have shown that the cochlea-palpebral reflex appears as a rule much earlier than the impediments to the sound conducting apapratus are supposed to have disappeared. Moreover, it has already been shown by Kutvirt and Gomperz that the late appearance of hearing in the newborn could be explained by the incomplete development of the inner ear and brain.

However, this explanation cannot be entirely satisfactory, since we know that the labyrinth as well as the brain of the newborn is morphologically completely developed, if we mean the structure in the neighborhood of the acustic fibres and center for hearing.

In cases of premature birth the conditions are naturally different and it is possible for the hypothesis of Gomperz and Kutvirt to be accepted in such cases. This is also true in cases of hereditary syphilis in the newborn.

In his second communication on this subject Kutvirt, as before mentioned, found a relationship of the reflex to the lengthened time of labor pain to the expulsion of the after birth.

He holds to the belief that in the newborn whose birth was associated with both protracted labor and late reflexes the head

in being held back was exposed thereby to increased pressure. I believe, however, that this obstetrical condition is not the most important cause in the delayed reflexes, for it must be remembered that, according to the statistics of Kutvirt in those cases which were delivered by Caesarian section after a long but tedious labor, the reflex appeared earlier than in those in whom the birth was only thereby retarded, as for example in breech presentation where the fetus was delivered by turning. From these circumstances it follows that the delay in the appearance of the reflex in the newborn in breech presentation was due to the increased compression on the skull in consequence of the increased labor pain. Moreover, infants who were delivered by Caesarian section had also suffered more from this greater compression. Therefore, we must the more, as I believe, attribute the late appearance of the reflexes in those cases of breech presentation to a longer and more continuous asphyxia of the after birth, and this must also cause some interference with the functions of the brain. We know, moreover, through Verworns, who has investigated the subject, that the brain is exceedingly sensitive to the withdrawal of oxygen.

From the above it is evident that Kutvirt, according to his observation of the newborn who suffer with a complicated labor and when the hearing reflex was late in developing, has only recognized the well known fact that the hearing reflexes can only be tested on the brain, possessing its complete function.

I have therefore undertaken to prove again these observations of Kutvirt.

Finally, it is well to note the abnormal changes following this reflex.

1. In two cases of normally born children of healthy parents the reflex was absent during the whole time while they were under observation.

2. In five cases of new born of syphilitic parents the reflex was likewise absent. It must be noted, however, that in the last mentioned cases the reflex appeared two to three days after the birth. On the other hand, I was able in one case to produce the reflex even 16 hours after birth. An earlier appearance of the reflex I was not able to observe.

3. In one infant where the mother suffered from eclampsia the reflex was able to be elicited for the first time three days after birth. This artificially delivered child had a body weight of 1800 gr.

4. In one child with scrofulosis which lived six days, no reflex could be obtained.

5. In one child born asphyxiated the reflex during the first two days could not be elicited.

6. In those of premature birth, i. e., seven or eight months, the reflex appeared six to eight days after birth, yet I was able in one premature birth, whose height was 43 cm. and weight 1805 gr., to obtain the reflex 15 hours after birth.

What especially concerns those cases of premature births is given in the following results:

Of the 18 premature births which had been studied, the reflex could be elicited in ten cases, between the sixth and ninth day after birth, in one each of the premature births; in the others, first on the 12th, 13th and 19th day after extrauterine life. Two of the premature births left the clinic on the eighth day and up to the time of departure no reflex was observed.

Of the two remaining premature births, the reflex appeared in one of them on the first day and this continued until the infant left the clinic. In the second case the reflex up to the 9th day of extrauterine life could be elicited and then it disappeared.

In all the cases with negative cochlea-palpebral reflex the appearance of the drum as well as the external meatus appeared normal by otoscopic examination. Among the premature births three children were found to show hereditary syphilis.

EXAMINATION HISTORIES OF THE PREMATURE BIRTHS.

No. 3607. Caroline Sp. History: Measles. Abortions 1906, 1908 and 1909. First pains began Dec. 17th, 3 o'clock a. m., birth 3:15 p. m. Length 48 cm., weight 2070 gr. Spontaneous birth. Reflexes appeared 16 hours after delivery.

No. 3681. Wilh. St. History: Rachitis. Pains began Dec. 23rd, 11 o'clock; birth at 4 p. m. Boy. Length 49 cm.,

weight 2520 gr. Spontaneous birth. Appearance of reflexes in 6 days.

No. 3451. Maller, Chr. History: Scarlet fever. Pains began Dec. 4th at 12 o'clock noon, birth 2:45 a. m. Boy. Length 48 cm., weight 2630 gr. Spontaneous birth. Reflexes 7 days after birth.

No. 3459. Leop. D. History: Measles. Pains began Dec. 6th, 2 o'clock a. m.; birth at 3:15 p. m. Girl. Length 48 cm., weight 2700 gr. Spontaneous birth. Reflexes 7 days after birth.

No. 3403. Josef Sal. History: Measles, pneumonia. Pains began Dec. 6th, 10 a. m.; birth 12:45 p. m. Girl. Length 50 cm., weight 2780 gr. Spontaneous birth. Reflexes 7 days after birth.

No. 3464. H. St. History: Grippe. Pains began Dec. 5th, 6:15 a. m.; birth 7 p. m. Girl. Length 46 cm., weight 2500 gr. Spontaneous birth. Reflexes 8 days after birth.

No. 3470. M. Ke. History: Measles, grippe. Pains began Dec. 16th, 9:30 p. m.; birth 11:15 p. m. Girl. Length 48 cm., weight 2700 gr. Spontaneous birth. Reflexes 8 days after birth.

No. 3486. Fr. R. History: For four weeks pleurisy. Pains began Dec. 8th, 12 o'clock midnight; birth 1:30 a. m. Girl. Length 47½ cm., weight 2700 gr. Spontaneous birth. Reflexes 8 days after birth.

No. 3487. J. G. History: Pains began Dec. 7th, 2 a. m.; birth 11:45 p. m. Boy. Length 47cm., weight 2450 gr. Spontaneous birth Reflexes 8 days after birth.

No. 3501. An. Sch. Pains began Dec. 7th, 11 p. m.; birth 12:30. Boy. Length 45 cm., weight 2130 gr. Spontaneous birth. Reflexes 9 days after birth

No. 3503. An. F. Pains began Dec. 7th, 7 p. m.; birth 9:15 a. m. Girl. Length 45 cm., weight 2260 gr. Spontaneous birth. Reflexes 9 days after birth.

No. 3509. Kat. Val. History: Measles. Pains began Dec. 8th, 11:30 p. m.; birth 2:30 a. m. Girl. Length 45 cm., weight 2680 gr. Spontaneous birth. Reflexes 12 days after birth.

No. 3511. An. Auf. History: Measles, asthma. Pains began Dec. 9th, 6 a. m.; birth 11 p. m. Length 48 cm., weight 2730 gr. Spontaneous birth. Reflexes 13 days after birth.

No. 3520. Ant. Fis. Pains began Dec. 9th, 3 a. m.; birth 1 p. m. Boy. Length 49 cm., weight 2800 gr. Spontaneous birth. Reflexes 19 days after birth.

No. 3523. T. Par. History: Bronchitis, grip. Pains began Dec. 10th, 3 a. m.; birth 4:45. Girl. Length 44 cm., weight 2000 gr. Spontaneous birth. Left the clinic on 8th day without any reflex having appeared.

No. 3531. P. L. Pains began Dec. 10th, 1 p. m.; birth 4:30 p. m. Girl. Length 46 cm., weight 2320 gr. Spontaneous birth. Left clinic on 8th day without reflex having made its appearance.

No. 3538. M. W. History. Abortions 1919. Pains began Dec. 12th, 2:40 a. m.; birth 1:15 p. m. Girl. Length 46 cm. Weight 2650 gr. Spontaneous birth. Reflex appeared on the 1st day after birth and after that disappeared.

No. 3545. V. P. History: Typhus. Pains began Dec. 12th, 11 a. m.; birth 1:30 p. m. Length 41 cm., weight 1850 gr. Spontaneous birth. Appearance of reflexes on 9th day after birth and again disappeared.

In order to understand why the hearing perception is absent in the perfectly healthy newborn we are obliged to fall back upon the functional conditions which have been indicated. For as we have mentioned before, every normal birth is associated with some degree of asphyxia in the infant, whereby a sudden change comes in the circulation of the new born, due to admission of oxygen. All of these conditions can be taken together to explain the decreased hearing ability of the brain in the newborn.

## CONCLUSIONS.

My investigations therefore lead me to the following conclusions:

1. The cochlea-palpebral reflex usually appears between the 4th and 6th hour after birth.

2. In premature births, say 6 to 8 lunar months, there is marked delay in the appearance of the reflex, as a rule, first between the 6th and 8th day ot extrauterine life.

However, there is sometimes an exception to this rule in that the reflex occasionally appears earlier than this (15 hours after birth) and sometimes later (12 to 19 days after birth).

3. In the hereditary syphilitic newborn, the reflex is sometimes normal and sometimes delayed.

4. Very seldom (2 cases out of the 105 observed) the reflex in those perfectly healthy and of normal birth is sometimes delayed as late as 20 days.

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LXVII.

# PRECAUTIONS IN USING LOCAL ANESTHESIA—REPORT OF A DEATH.

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The study of the susceptible patient and the method of administration present the most interesting and complex problems in the use of local anesthetics. For the average individual the moderate dosage of a recognized local anesthesia has little danger. It should not be overlooked, in the daily rush of our routine, that even the mildest of these drugs are highly toxic and that there are more than a few individuals who possess an idiosyncrasy for them.

Inasmuch as practically all drugs used for local anesthesia produce almost identical toxic symptoms, novocain or procain, the least toxic of those in general use, will be referred to unless otherwise mentioned.

After the consideration of the hypersusceptible patient the next most important question is the method of administration.

Susceptibility.—Of all the drugs to which a patient may think he is susceptible, his statements in reference to cocain and allied drugs should be considered most reliable. Individuals who have been poisoned by cocain or procain are more susceptible to the second administration. If no history of having had the drug used before is obtained, susceptibility of the patient should always be tested by using a small, well diluted dose previous to giving the full amount necessary for anesthesia. For example, when procain is to be injected for the removal of tonsils, it is my practice to inject about ½ c. c. of a ¼ of 1 per cent solution at a point to be used later; after waiting five minutes, if no symptoms are produced, to proceed with the full amount. Similar precautions should be taken before any injection or before applying large amounts of strong solutions of cocain to the mucous membrane.

Administration.—The guiding fact should be first to keep well within the limits of safety in the amount of the drug

used. The dose of procain or novocain as given by different authors varies widely. Roth,[1] after quoting from twenty-one authors, states: "From the above references no definite conclusions can be drawn as to the exact toxicity of novocain in man. Under certain conditions 0.02 gm. (approximately ⅓ of a gr.) has caused poisoning symptoms, while as much as 2 gr. (approximately 15 grains) has been given without producing symptoms." He further states: "The untoward results that have been reported in the literature from the use of novocain in operations about the head and face might well be accounted for by the fact that absorption may be rapid." "Individual susceptibility is marked in both animals and man."

The dose of cocain has also wide limitations according to the authorities; ½ to ¾ grain is usually considered safe.

To keep well within the safety limit, 2 to 3 grains of procain and ½ to ¾ grain of cocain can be used in nose and throat surgery.

In the susceptible patient much smaller doses may cause serious toxemia, while in the nonsusceptible individual large doses are safe if guarded by measures to reduce the rapidity of absorption. This can best be accomplished by injecting or applying the drug in only small quantities at a time combined with adrenalin solution or allied drugs which cause vasoconstriction.

Allen[2] in his book on Local Anesthesia states: "It is believed that cocain once fixed by the body cells (in combinations with them) is not liberated from these combinations as cocain, but as constituent products, ecgonin, etc.; Glasenap believes he has isolated ecgonin from the urine. These derivatives of cocain are slightly anesthetic and slightly toxic but much less so than cocain. As a result of the preceding statements it can be said that cocain exerts its full anesthetic or toxic action but once, and if exhausted locally there will be no constitutional reaction."

For injective anesthesia, the smallest amount of the least toxic drug which produces the desired result should be injected slowly, changing the position of the point of the needle after each few drops are injected, thus avoiding the danger of inducing any great amount directly into the circulation should the needle at any time enter a vessel. Owing to the great

vascularity of the tissue surrounding the tonsil, massive single doses injected in one place for anesthesia should be condemned, as should also large cotton tampons hypersaturated with a strong solution of cocain placed in the nasal cavity and left there for a long time. In the applications to the nose and throat precaution should be used to guard against the dropping or running of the solution to other parts.

One should be constantly on watch for the first toxic symptoms, and, since they often occur suddenly and when least expected, should guard himself by the knowledge of how best to combat them.

The first toxic symptoms of cocain are usually those of excitation—talkativeness, laughing, nervous fear, etc. This stage of excitation may be absent, and I believe usually is, when procain or novocain has been used. Nausea, pallor, sweating, pain in the head, faintness and collapse, combined with rapid pulse and a feeling of constriction of the chest, may be seen as early symptoms.

It is a mistake to attribute such symptoms to the nervousness of the patient. They may suddenly terminate in convulsions and death. In severe toxemia, convulsions simulate epilepsy. Beginning with the head and neck, followed by general tremor, short, jerky spasms involve the entire body, are accompanied with cyanosis, tightly clenched teeth with frothy mucus exuding from the mouth, rapid weak pulse, and rapid labored respirations—the pupils may or may not be dilated. The convulsion lasts for a few minutes, but after subsiding for a short time may be followed by others and death.

Treatment.—If the patient is not already in the reclining position he should be placed so at once. Should the state of collapse continue heart stimulants should be administered hyperdermically and inhalations of amyl nitrate or ammonia given. For the convulsive movements, although not tried by the author, inhalations of ether would seem to be by far the best. Artificial respiration and massage of the heart should be practiced.

Quoting from Allen:[3] "For nervous excitement or convulsions, H. C. Wood recommends chloroform by inhalations, but it would appear that ether should be better, particularly if it be proved that the central action is associated with the

same vasoconstriction and anemia that takes place in its local field of action; inhalations of ether, due to the tremendous congestion which it produces in these parts, should prove of graet value, besides stimulating vasomotors, heart and respiration and controlling the convulsions; nitrous oxid would be equally as valuable; amyl nitrate, while producing the same congesting effect, would not exert the same controlling influence upon the convulsions.

Engstady[4] says, in speaking of cocain poisoning: "In the first few cases I was called on to treat, strychnin and morphin in combination were used with marked benefit. But, as cases kept multiplying, I found the action of these drugs too slow, and I decided that there must be something to counteract the poison more rapidly when life was in extreme danger. It was necessary to find a remedy that could be administered at any time and be instantaneous in its action. I soon found ether to be the required drug. This was administered as ordinarily given to produce surgical narcosis. Ether stimulates the vasomotor system, is a tonic to the heart muscles, stimulates the action of the respiratory centers of the brain and of the pneumogastric nerve, and increases the pulmonary circulation in the first stages. While cocain inhibits the action of the heart, especially on the right side, it has also a marked inhibitory action on the respiratory centers of the brain. Death may occur from feeble respiratory movements of the socalled Cheyne-Stokes type or asphyxia.

"To me ether has proved extremely valuable. It has saved what seemed hopeless cases. It stimulates the heart and the respiratory system almost instantly. The pulse becomes fuller at once and of normal tension. The marked mental excitement is allayed as the poison rapidly disappears. The individual regains consciousness as soon as the effect of the small amount of ether has disappeared."

Allen[5] further advises the use of opium and its alkaloids. He believes, however, it is best used as a prophylactic or preventive by the preliminary use before the injection of the anesthetic "when it does certainly permit the use of much larger doses of the anesthetic and in this way acts as a direct antagonist."

Allen[6] does not think the injections of adrenalin advisable after the toxic symptoms. He believes they are caused by paralysis of the higher nerve centers, adrenalin exerting the same action upon these centers as it does locally, causes a prolonged retention of the toxins in the brain.

On the other hand, Eggleston and Hatcher[7] and Braun[8] advise the infusion of epinephrin solution upon the recognition of the first toxic symptoms to stimulate the action of the heart so that the toxic agent may be brought in contact with the liver, where Eggleston and Hatcher believe the toxins are destroyed, quoting from Eggleston and Hatcher:[7] "Since acute poisoning in man occurs where least expected, those who frequently employ local anesthetics should always be prepared to apply the three resuscitative measures of intervenous infusion of epinephrin, artificial respiration and cardiac massage. All three should be used in combination, since no one is efficient, except in rare instances." They base their belief upon extensive experiments upon cats; they were able to resuscitate cats after a rapid intervenous injection of doses of local anesthetics up to twice the usual fatal dose by using the above measures.

All recent articles written upon this subject claim that these drugs kill by paralysis of the heart and respiratory centers.

The committee appointed by the Otolaryngologic Section of the A. M. A., to study the "Advantages and Disadvantages of Various Local Anesthetics in Nose and Thorat Work,"[8] after two years' study and communication with the members of the otolaryngologic section, report forty-seven deaths from local anesthesia; all except eight cases were tonsil cases. Of the cocain deaths it is appalling to note that one-fourth were due to "mistakes of druggist or nurse."

After carefully selecting the tonsil cases which had no contributing causes, such as tuberculosis, heart disease, mistakes, etc., reported, I found nine deaths caused from injections of cocain ranging in strength from 0.2 per cent to 3 per cent. Twelve deaths resulted from injections of procain solutions ranging in strength from 0.5 per cent to 2 per cent. In thirteen of these epinephrin or adrenalin was used in the solution. In the procain cases eight had concain applied to the pharyn-

gical surface before injection. Four had only procain injections.

The greater number of procain deaths in these selected cases occurred because of better reports given of them. In the full number of forty-seven, twenty-five were cocain deaths, fourteen were procain deaths. The other deaths were: One, from the use of alypin, which is very toxic, and two from apothesin. Morphin injections had been given to approximately half of the cases. Procain or cocain was the only constant medication.

Becker[10] reports a death of a girl 25 years old following an injection of about 100 minims of 1 per cent novocain for the removal of tonsils. The patient died suddenly with symptoms simulating an epileptic convulsion. A larger amount of solution from the same container had been used upon a sister in the same manner without any toxic symptoms.

Simpson[11] mentions a death of a male patient before a tonsil operation had been begun. Hypodermic morphin, gr. 1/6, was given one-half hour before, followed by local application of 10 per cent cocain solution fifteen minutes before. While in the act of injecting ½ of 1 per cent novocain the patient was suddenly attacked with what Dr. Simpson thought was an epileptic seizure, followed in a short succession by six or eight more convulsions, and died shortly after.

White[12] reports a tonsil case which was being prepared for operation in his clinic. "The patient, a woman, aged 23, rather heavy set, was prepared for local tonsillectomy without the previous use of morphin as follows: The tonsils, pillars and pharynx were swabbed with a 20 per cent solution of cocain with 10 to 15 drops of 1/1,000 adrenalin to the ounce. This was followed in about ten minutes by the injection of a 1 per cent solution of novocain. About three drams of the solution was drawn up into the ordinary type of syringe used for this purpose, and the injection was made in the usual sites on the right side and about the same amount was injected on the left side. About fifteen seconds after the last injection the patient with an exclamation reeled forward in convulsions. Cyanosis was marked. The patient was laid flat on her back on the floor, the sphincter ani dilated and artificial respiration begun, but the convulsions persisted and cyanosis was still very marked; the pupils were semidilated, the pulse very faint, and

the respirations shallow. The heart was massaged, ether and adrenalin given subcutaneously, and the use of the pulmotor was instituted. The convulsions ceased, no respiration was evident and there was no perceptible heart beat or pulse. The artificial respiration was continued for about an hour, at the end of which time the patient was pronounced dead. There was no apparent cause for the condition. At the postmortem all the organs were found normal, excepting the thymus, which weighed about 50 grammes. Diagnosis: Status lymphaticus."

After operating upon nearly 1,000 tonsil cases under local anesthesia without any serious symptoms, a patient I was carefully preparing for operation before a public clinic suddenly died before the operation had been begun. On March 15, 1921, R. A., a young man, 18 years old, blond, of medium build, with rather effeminate features, was prepared for operation as follows: Morphin sulp., gr. 1.4, with atrophin sulp., gr. 1/150, had been given twenty minutes previously. Ten per cent solution of cocain was applied to the superior and inferior poll of each tonsil by placing a cotton tipped applicator saturated with the solution for a few seconds in each place.

Following this about 1½ drams of 1 per cent procain solution containing 1 drop of adrenalin, solution 1/1,000 to the dram (approximately 1 1/5 grains of procain plus 2 drops of adrenalin solution), was injected outside of the capsule on each side. In about one minute after the last injection, while waiting for the anesthesia to take effect, the patient said he felt badly and looked very pale. His head was placed between his knees for perhaps another minute, when he began with a slight convulsive movement involving the head and upper part of the body. He was at once placed upon a stretcher, by which time the convulsive movements involved the whole body. Short, rapid, jerky spasm, lifting the extended legs and head and shoulders from the stretcher, was accompanied by a general tremor, marked cyanosis, eyes open, tightly clenched teeth, with biting of the tongue and frothy bloody mucus exuding from the mouth. The pulse was weak and rapid, becoming almost imperceptible.

The respirations were shallow and labored. After lasting one or two minutes the convulsions subsided to a general tremor, with improvement of the pulse.

A second convulsion followed, after which the patient was taken to the ward, where the pulmotor and other measures were used to resuscitate him without avail. He died in about thirty minutes.

Solution from the same bottle had been used for a patient just previous to this one without toxic symptoms.

Having been told before the clinic began that one of the patients to be operated upon was subject to epilepsy, I at first mistook the symptoms for an epileptic seizure.

The autopsy showed a much enlarged spleen and general hypertrophy of the lymphoid tissue throughout the body. The postmortem diagnosis was "status lymphaticus." The fact remains that the patient died with symptoms of novocain poisoning.

To almost every laryngologist there comes a period of enthusiasm for the use of local anesthesia in tonsillectomy. To operate upon a conscious patient without pain or bleeding and to have a grateful patient, grateful especially because of the elimination of the fear of ether and its after effects, is encouraging; add to this a more or less lengthy series of such cases, anesthetized with a comparatively small amount of a solution of less strength than the general surgeon uses in almost limitless quantities—and the enthusiasm of the young laryngologist is apt to relax his vigilance and to cause him to select too wide a field for the use of local anesthesia.

It needs to be constantly borne in mind that ether, except in selected cases, is the safer anesthesia.

There is a large number of selected cases in which local anesthesia is the method of choice. This is especially true in operations upon the nasal cavity, in pulmonary, renal, hepatic disease and in the aged.

Local anesthesia is especially contraindicated when operating upon the nose or throat of children, highly nervous or neurotic subjects, in epileptic and static lymphatic cases and in some cardiac conditions.

SUMMARIZING THE PRECAUTIONS IN USING LOCAL ANESTHESIA.

1. A careful history and physical examination of the patient, especially to detect susceptibility, neurosis, epilepsy, cardiac lesions and the existence of a lymphoid diathesis.

2. Elimination of the patient's susceptibility to the drug used.

3. The use of the smallest amount of the least toxic drug well diluted which will produce anesthesia.

4. The addition of adrenalin or epinephrin to all solutions to reduce the rapidity of absorption.

5. The solutions should always be administered slowly. When injections are given every care should be taken not to enter a vessel.

6. The reclining position is to be preferred, thus relieving all strain from the heart.

7. It should be the infallible rule of every surgeon to freshly mix his own solutions and to keep the stronger solutions for surface application in a different colored container.

8. The operator should be prepared at all times to administer the measures to combat toxic symptoms.

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## LXVIII.

# THE WARING SUCTION TONSILLECTOMY.

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In recent years, especially perhaps in the past decade, the tonsil question has loomed larger and larger to the medical profession, and in common with the laity, to a large extent has arisen the idea that the tonsil affords the chief gateway for entrance of various systemic infections. Increased knowledge of pathology in general, and especially the development of the focal infection theory, have served to place the tonsil in the medical limelight more and more.

Tonsillectomy has become a very common operative procedure with the growing demand for removal of tonsils in adults afflicted with this and that ailment. Conservatism is in the air, however, and it is needless to caution against indiscriminate removal of the tonsils, and to emphasize that tonsillectomy should not be resorted to until all other likely etiologic factors have been eliminated in the given case.

I think it is universally admitted that complete removal of the tonsil, including the capsule, the socalled radical tonsil operation, is the operation of choice on the part of the great majority of American rhinologists, in the belief that unless the entire tonsil is removed full benefits may not be secured.

It is true that some rhinologists do not favor removal of the tonsillar capsule, but this view is not held by the majority, and removal of the capsule certainly better insures total ablation of the tonsil and greatly facilitates operative technic.

It is unfortunate that there has survived from the older "clipping," or partial removal tonsillectomy, a rather widespread impression that there is little danger in removal of tonsils, and that it is an operation to be performed with impunity anywhere and by any physician. We should strive to correct this impression and have it more generally realized both by the profession and by the laity that tonsillectomy is more of a major surgical procedure, not unattended with danger, under

even the most favorable conditions, and an operation to be performed as far as possible in hospital or well equipped office only and by trained rhinologists.

As the case stands today, however, tonsillectomy is performed not only by the trained throat specialist, but almost as commonly by the general surgeon; by the general practitioner with surgical leanings, and also by the recent graduate ambitious to "rise in his profession." Tonsillectomy today is performed in hospital, in office and in the home; with assistants and without; under general anesthesia and under local anesthesia.

Tonsillectomy technics today may roughly be divided into two main types, namely, some form of snare and dissection and some form or modification of the ingenious method brought out by Sluder in 1910.

The innumerable variations of this and that technic, together with the multitude of tonsillotomes, tongue depressors, retractors, tonsil hooks and tenaculums, knives, scissors, punches—all serve to indicate that the last word has not been reached in tonsillectomy technic; that the methods in use today offer certain dissatisfactions in end-result or difficulties in operation which lead the earnest operator of mechanical turn to strive to improve his armamentarium and his technic.

Some form of snare and dissection is perhaps most used today, and in the hands of experts and on tonsils of appropriate size and type gives very satisfactory result. In the hands of the average operator, however, snare and dissection technics usually leave considerable to be desired. The operation is often tedious and time consuming, and a strain on the patience both of the patient and operator under local; and under general anesthesia often means much time lost under anesthesia. Dissection work is often bloody and, with a field obscured by blood and mucus, most difficult and trying. Advent of suction pump apparatus has been a great advantage in clearance of the operative field, but even with this, dissection work on badly adherent and submerged tonsils often degenerates into a mere cutting out procedure instead of orderly dissection. There is often more or less damage to pillars and laceration of throat muscles, so that convalescence is often

delayed, stormy and painful. On friable and ragged tonsils it is often difficult or impossible to obtain a satisfactory grasp with any of the numerous tonsil seizing forceps, screws or tenaculums; or when a satisfactory grasp is obtained most annoying and delaying to have the instrument pull out of the tonsil at a critical stage, with attendant shredding of the remaining tonsil tissue. More or less free bleeding is often met with in dissection work, and aside from the weakening effect of loss of blood, much valuable time is often lost in efforts at hemostasis. The extensive armamentarium usually at hand for this type of tonsil operation likewise has a rather disconcerting effect when viewed by the patient under local. Often again snare and dissection means repeated snaring of bits of tonsil tissue left—more labor and more time consumed.

Turning to the ingenious technic of Sluder with its various modifications, a few of its disadvantages may be pointed out briefly in passing. Advantages claimed for it are rapidity, minimum of surgical violence and less hemorrhage.

In the hands of experts in its use, the Sluder type operation is almost spectacular in its comparative rapidity and clean end result, but it is not a technic applicable to all forms of tonsils, and the Sluder operator soon learns to recognize the tonsils he cannot "get" satisfactorily with this technic, and turns to some other secondary mode of attack. Usually the tonsils hard to "get" with the Sluder technic are the small, deeply submerged and adherent tonsils, so often badly diseased and in need of removal. Quite often one or both pillars are damaged or even completely removed by the Sluder method, and with the socalled sharp Sluder free hemorrhage often follows the enucleation. With the dull or two bladed type of Sluder instrument, such as the LaForce, Beck, Meding, etc., a practically bloodless enucleation may be done, and on appropriate type tonsils a very satisfactory operation. However, the instrumental manipulation called for by this technic is often quite difficult for the average man to acquire satisfactorily, and its successful performance calls for a considerable degree of "strong arm" muscular accompaniment, which all operators do not seem to possess. Likewise visibility at certain stages of the manipulation is rather obstructed, with instrument and fingers in mouth and throat at same time.

It has been my intention to point out briefly in passing such disadvantages of the two main type tonsillectomy operations in use today as might appear to the average operator and not to decry in any way the hundreds of brilliant operative results secured by expert operators every day by these two technics.

It has been with an open mind, but faced with the difficulties and disadvantages of modern tonsil work, that the subject of tonsillectomy has been approached by the writer from what may be termed a new or a modified angle, in an effort to perfect a tonsillectomy technic which would be equally applicable to all types of tonsils; be rapid, clean and bloodless, and likewise insure good tonsil work in the hands of the beginner and the occasional operator, no less than in the hands of the average and the expert operator.

In a considerable use of tonsil suction tubes for diagnostic and therapeutic purposes, I was impressed not only with the thorough mechanical "vacuum cleaner" cleansing of tonsils by suction, but equally so with the ease and completeness with which even deeply submerged tonsils were drawn out of their beds and held firmly in the mouth of the glass suction tube while the suction pump was turned on.

Pus and masses of caseous and ill smelling purulent exudate were drawn out of the tonsil crypts and into the suction tube, thus mechanically removing from the tonsil and from the throat much potentially infective material, which if left and swallowed or reabsorbed could well cause considerable trouble.

In the opinion of the writer, this "vacuum cleaner" mechanical cleansing of the tonsils prior to actual operative attack can well be incorporated as a preliminary step in tonsillectomy technic, applicable to any and to all tonsil removal operations.

As one confrere wrote me recently: "It occurred to me how easy it would be to throw a snare loop over the tonsil already almost turned inside out by grasp of the suction tube and thus enucleate." Using an appropriate size glass suction tube, it was found that the tonsils were drawn out of their beds and held in the mouth of the suction tubes far better than with any of the numerous tonsil grasping forceps, and held there practically inverted as long as suction was on. Even without anesthesia of any sort patients complained of but little dis-

comfort during the maneuver, and there was no laceration or shredding of tonsil tissue as forceps so often entail. As desired, however, the suction grasp on the tonsil could be instantly released at will by disconnection of the suction pump and as easily regrasped by suction.

High visibility was another feature that appealed to me. One can see exactly what he is doing, as the clear glass of the suction tube offers little obstruction to vision.

With the first few enucleations attempted with the snare and suction tube, a plain glass tonsil suction tube of the Hurd type was employed, but it was found this was not satisfactory, because in a certain percentage of cases the snare loop would hang up on the lip of the tube and as tightened down slide down towards the shaft of the tube instead of on to the base of the tonsil to be enucleated. This necessitated release of the tonsil, readjustment of snare and a fresh start. In the cases where the snare loop did slide down over the base of the tonsil it was found enucleation could be performed rapidly, cleanly and in a practically bloodless manner. The tonsil was removed cleanly in capsule, with no damage to pillars, uvula or other throat structures; the fossæ left clean and convalescence rapid and uneventful.

To overcome the only mechanical imperfection met in the procedure I have designed a new type of tonsil suction tube, all glass with rubber handle for ease of manipulation, and in a set of varying sizes to accommodate all sizes and types of tonsil. These tubes have a rather bulbous head set at about right angles to the shaft of the tube, short necked and with smooth shoulders sloping rapidly down to the aperture, which is broadly oval in its perpendicular axis.

With these improved suction tubes the operation may be said to be 100 per cent perfect in execution and in end result. Practically all physicians doing throat work at all have some form of compressed air and suction apparatus in use, and any of the modern suction pump apparatus should give sufficient suction for this operation.

Where general anesthesia is employed, a small vacuum bottle of from ½ to 1 pint capacity may be incorporated in the rubber tubing vacuum circuit back to suction pump, so that as needed the throat may be cleansed of mucus and saliva,

using the same glass suction tube as for enucleation, but merely turning same with aperture down into the pharynx. Owing to its rapidity and bloodless type of operation, only a very brief and light general anesthesia is required, as little or no time under anesthesia is expended in efforts at hemostasis.

Where local anesthesia is employed, the tonsillar region and pharynx may be anesthetized by whatever method the individual operator prefers. The operation is essentially a one-man job and may be employed equally well in hospital or office and, except for an anesthetist where general anesthesia is employed, no other assistance is required in the operation.

Any type of tonsil snare may be employed, although in the opinion of the writer snares having a ratchet mechanism, such as on the Beck-Pierce and similar snares, offer certain advantages in a more nearly bloodless enucleation. About a No. 8 snare wire gives good results. As a rule the snare and suction tube are the only instruments required, the suction tube being used somewhat as a tongue depressor as it is carried into the mouth and over the tonsil to be enucleated.

An appropriate sized Waring suction tube having been connected up to the suction pump by about four feet of flexible rubber tubing, the snare loop is now passed over the suction tube and allowed to hang freely suspended out of the way until required. The suction tube should be of a size sufficient to firmly grasp and encircle the tonsil to be removed. Under direct inspection the suction tube is carried into the mouth and its mouth carried between the pillars and over the tonsil to be removed. Suction is now turned on, when it will be seen through the clear glass of the suction tube that the tonsil is drawn completely out of its bed, almost inverted, and firmly held in the grasp of the suction tube. High visibility is one of the most appealing features of the operation to me; at every stage you can see exactly what you are doing. There is no danger of including the uvula or damaging the pillars by a grope in the dark. Likewise success of the operation does not rest on heavy muscular exertion or difficult instrumental manipulation in the throat; the operation is simplicity itself, as will be seen at a glance from the cut. There is no preliminary dissection, no employment of knife, scissors, retractors, tonsil seizing forceps, etc.

As the tonsil is grasped by suction the snare loop is now carried over the bulbous end of the suction tube, and on to the base of the tonsil, the snare being grasped by the left hand for the right tonsil and by the right hand for the left tonsil. As the snare loop is tightened down, the smooth, sloping shoulder of the suction tube automatically carries it down and over the base of the tonsil to be enucleated. At this stage the tonsil may be enucleated rapidly; or, if desired, enucleated slowly by a gradual screwing down of the snare loop, as is possible with the Beck and similar type snares. This gives a more bloodless operation, but is not essential. The operation will be found practically bloodless in all cases, the rather heavy snare wire crushing rather than cutting through tonsillar vessels as the base of the tonsil is separated from its bed.

It will be found the tonsil is practically inverted and cleanly enucleated in capsule, fossæ left smooth and no damage to pillars or other throat structures. There is no cutting or laceration of the pharyngeal muscles, as often occurs in dissection work, and as a result a minimum of postoperative sore throat is complained of, and convalescence very rapid and uneventful; the majority of patients eat their regular meals the second morning. Owing to its rapidity and bloodless nature there is practically little shock, even in patients comparatively advanced in years.

The second tonsil is removed in the same manner. Any oozing in fossæ may be very effectually controlled by a swab of Mousel's solution in glycerin, but this is rarely needed, and secondary hemorrhage after this technic has not been encountered in any case.

Advantages of the suction tonsillectomy over preexisting technics are briefly as follows:

The operation is so simple that even the beginner and occasional operator can do good, clean, bloodless tonsil work with the assurance of uniformly good end results, and do it in a manner more rapid and more satisfying both to patient and to operator than with the older technics.

The operation is practically bloodless in all cases, so that no time is lost in efforts at hemostasis. Where general anesthesia is employed, its rapidity and clean type calls for only a very brief and light anesthesia, so no time is lost in hemostasis.

The operation is essentially a one man job and does not require the train of assistants and extensive instrumental armamentarium usually met with in tonsil work.

High visibility is another strong feature of the operation. Every step of the operation is under direct inspection, and there is no difficult instrumental manipulation inside the mouth as with Sluder type operations. The technic is simplicity itself.

There is no type of tonsil the operator cannot "get" easily, rapidly and cleanly by this operation, and its superiority will quickly be demonstrated on the small, deeply submerged and adherent tonsils so difficult of clean attack under ordinary methods.

On account of its clean type, smooth fossæ and lack of damage to adjacent throat structures, very little postoperative sore throat is experienced and convalescence will be found rapid and uneventful.

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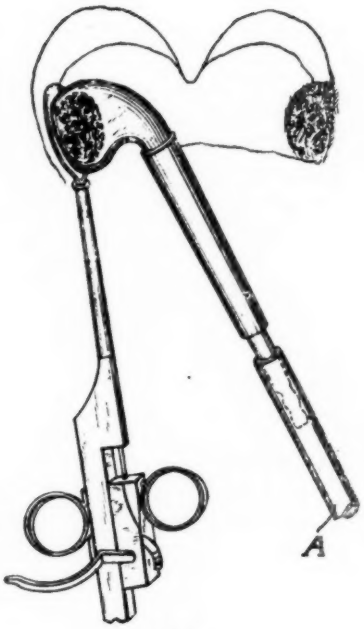


FIGURE 1.

The right tonsil is engaged in the bulbous mouth of a Waring suction tube, which is connected by rubber tubing "A" to suction pump. Snare loop has automatically slipped down over sloping shoulders of suction tube in readiness to enucleate. Beck-Pierce snare in use in illustration, but any type of tonsil snare applicable.

LXIX.

# OTOGENIC SYMPATHETIC MENINGITIS—REPORT OF A CASE WITH RECOVERY AFTER A RADICAL MASTOID OPERATION.

By Jesse Wright Downey, Jr., M. D.,
F. A. Pacienza, M. D., and Oscar G. Costa, M. D.,
house staff, mercy hospital,
Baltimore.

The following case record is a striking example of the variously called sympathetic, protective or serous meningitis, due to a chronic suppurative otitis media presenting classical symptoms, typical laboratory findings and a surgical demonstrable area of meningeal irritation.

It is undoubtedly of the class in which the great majority of reported recoveries from otogenic suppurative meningitis probably belong, and it substantiates the experiences and opinions of Eagleton, Yerger and others that meningitis of otitic origin may be amenable to surgical treatment so long as it remains in protective stage, which is indicated by a sterile spinal fluid.

Jos. P. White, age 49, was admitted to the Mercy Hospital May 20, 1922. He had been previously treated by Dr. M. Raskin for a chronic suppurative otitis media of twelve years' duration, which had recently increased in severity with periodic manifestations of severe pain, headache, vertigo and unconscious spells, which lasted from three to five minutes and had been called epilepsy. The left ear had shown a large polypoid mass, whose atypical appearance suggested malignancy and justified removal for microscopic examination. A portion of the polyp was carefully removed by Dr. Raskin on May 18th, and within forty-eight hours the man returned almost frenzied with headache, mentally confused, and evidently very ill. He was immediately sent into the hospital, where Dr. F. A. Pacienza, Dr. O. G. Costa and I examined him shortly after his admission, finding the following diagnostic symptoms:

Patient poorly nourished, semiconscious, moaning with pain in head, restless and irritable. Questions indefinitely answered. Temperature, 102. Pulse, 120. Respiration, 26.

Indefinite paresis of left external rectus. Pupils and eye-grounds normal.

Profuse offensive purulent discharge from left ear. Drum absent, tympanum filled with granulations. Left mastoid and parietal regions tender on palpation.

Neck and back muscles rigid; patient showing definite opisthotonos. Kernig's sign positive. Brudzinski's sign positive. Reflexes exaggerated. Slight spontaneous tremor of arms and legs. Heart, lungs and abdomen negative.

There could be but little doubt that we were dealing with a septic meningitis of otitic origin, and this impression was confirmed by a spinal puncture. The spinal fluid was under marked pressure, creamy in appearance, and the following laboratory report was given: Cells, 6,000 to cu. m. m., 96 per cent polymorphonuclears, 4 per cent lymphocytes. Globulin positive. Wassermann negative. No microorganisms found in stained slides made from a centrifugalized specimen.

The blood count showed a leucocytosis of 19,900, with 92 per cent polymorphonuclears, 5 per cent small mononuclears and 3 per cent mononuclear cells.

The absence of bacteria in the spinal fluid, in conjunction with a chronic suppurative otitis media, now exhibiting the signs of an acute exacerbation, made me feel that an exploratory radical mastoid exenteration was justifiable. It was impossible, however, to get the consent of the patient's family until his wife, who was out of the city, could arrive. This meant a delay of another twenty-four hours. In the meanwhile the man's condition remained unchanged and cultures made on blood agar from the spinal fluid remained sterile. This laboratory report almost assured us that we were dealing with a socalled protective meningitis, and the chances of success from a surgical procedure were, therefore, somewhat better. Accordingly on May 22d, a mastoidotympanal exenteration was performed.

The cortex was extremely hard, but the cellular structure of the bone was not entirely sclerosed and a softened spot was found in the upper posterior region of the mastoid which, when curetted away, disclosed an absence of the bony wall of the sinus over an area at least a centimeter in diameter. The edges of this opening were smooth and the surrounding bone

sclerotic, making it evident that the bone destruction was of long standing.

The exposed dura was covered with small vascular granulations and was bound by adhesions around the whole circumference of the exposed area. We were very careful to avoid removing or breaking down any of these dural granulations or adhesions.

The mastoid antrum was deep, of average size, and filled with pus, granulations and cholesteatomatous material, with an odor indescribably offensive. The dura was not found exposed in this region and the roof of the antrum and tympanum appeared healthy.

The plastic part of the operation was omitted and the wound left open and packed with iodoform gauze.

Cultures made from the mastoid at the time of operation remained sterile. It is interesting to note, however, that these cultures were made from the region of the dural exposure and not from the foul secretion found in the antrum.

On May 23rd, the patient's mental condition showed marked improvement. He was more comfortable and answered questions fairly accurately. Maximum temperature, 101; pulse, 100.

On May 24th, his condition was markedly improved. Temperature, 99; pulse, 90. Very little headache. A spinal puncture was made, which showed the fluid under but slight pressure and but faintly cloudy. A count of the cells showed 90 to the cu. mm., and the differential count showed polymorphonuclears 8.5 per cent, small mononuclears 86.5 per cent, large mononuclears 5 per cent. The wound was draining freely and very foul.

On May 26th, the temperature was normal. Another spinal puncture gave a clear fluid under no pressure. Globulin still positive.

On May 28th, spinal puncture, fluid clear, no pressure, globulin negative, and no cells.

On June 26th, the man was discharged from the hospital, mentally clear, and in good physical condition.

When last seen (October 9, 1922) he was back at work, apparently in good health, free of headache and his socalled epileptic attacks.

## SOCIETY PROCEEDINGS.

### CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Meeting of Monday Evening, March 5, 1923.*

THE PRESIDENT, DR. C. H. LONG, IN THE CHAIR.

**Paper: "Parotid Fistulæ in Mastoid Operations."***

BY WILLIAM E. GROVE, M. D.

MILWAUKEE.

DISCUSSION.

DR. JOSEPH C. BECK said he never had encountered such an unfortunate condition as a double fistula of the parotid gland. He did not think it likely that the Doctor's case was the result of injury by a sharp instrument on both sides. The only experience Dr. Beck had had with a parotid fistula was where a nerve anastomosis between the facial and spinal accessory nerves was performed. In that specific case the assistant penetrated the parotid gland with a sharp retractor, and Dr. Beck called his attention to the probability of a parotid fistula resulting from that accident. It did, and continued to discharge for several weeks. The introduction of a conical point which was charged with electric heat (surgical diathermia) finally succeeded in closing the fistula. No doubt this procedure resulted in the coagulation of the epithelium lining the fistula and allowed cicatrization to take place. This case was reported in connection with a few cases that were operated on for anastomosis. The facial paralysis, however, did not recover.

DR. J. HOLINGER reported an experience with a parotid fistula in the three-year-old girl of a colleague. The abscess of the parotid started one week after operation. The parotid was not injured during the operation. In order to avoid a scar on the face later he incised the abscess subcutaneously from the mastoid wound. He advised the parents to omit all fruit from the diet, but the father thought this was a "grandmother

*See page 855.

fad" and paid no attention to it. The abscess persisted and necessitated daily changes of dressings for six weeks. Dr. Holinger was convinced that the fruit acid caused an increase in the secretion of saliva and persuaded the father to leave off all fruit and fruit juices, whereupon the fistula closed in two days.

Dr. George E. Shambaugh said that study of the anatomy of the external meatus has disclosed a condition which permits of extension of infection from the parotid into the external meatus and of an infection in the walls of the canal through to the parotid. The anterior wall of the external part of the canal is formed by a wedge shaped tongue of cartilage which has two clefts called the incisura of Santorini. These clefts are necessary for the movement of the auricle in the lower animals. It is through these incisura that the infection extends. Dr. Shambaugh had never seen a case where a furuncle had broken through the meatus into the parotid, but he had seen an abscess of the parotid which broke through into the external meatus.

Dr. Otto Stein said he had had no experience with parotid fistulæ in connection with mastoids. He had seen an abscess of the parotid break through into the canal followed by a fistula and had seen one case of fistula in the parotid canal due to infection from opening a furuncle, but he had never seen such a case as that reported by Dr. Grove.

Dr. W. E. Grove (closing) said that when the first of the fistulæ occurred he was willing to plead guilty to having been clumsy in his operative procedures and possibly to having injured the parotid gland in some way, but when he operated on the child on the opposite side with the first experience in mind, he was certain he did not injure the parotid gland. His only explanation for the thing occurring on both sides was that there must have been a much displaced parotid gland which projected backward into the field of operation.

He agreed with Dr. Holinger that with a salivary fistula of this kind it would be wise to avoid the use of any foods that would stimulate the flow of salivary secretion. The more a fistula can be kept from discharging the better chance there is for prompt healing.

**Paper: "Experiments on Overirritation of the Labyrinth (at Professor Siebenmann's Clinic of Basel, Switzerland)."**

By J. Holinger, M. D.

ABSTRACT.

The essayist briefly reviewed a long series of experiments that have been published under several names in the Zeitschr. f. Ohren., and the special work by N. Satah, and presented lantern slides which demonstrated this interesting work in detail.

He stated that the middle ear was always found free from changes if there had not been any intentional interference, such as removal of the anvil. The vestibulum and the semicircular canals were also free from change. The cochlear nerve, the spiral ganglion and the acoustic nerve were free in the beginning but became successively atrophic with the progress of the atrophy of the respective parts of the organ of Corti.

These experiments showed that bone conduction does not influence the result of overirritation. In the human there is increased bone conduction in disease of the middle ear, but if this were as important as Wittmaack believes, the ears without the anvil could not show an intact organ of Corti. Hoesslin and Satah concur in the opinion that the membrane of Corti is the active part, and this corresponds with the findings of Dr. Shambaugh in the ampullæ, where a similar organ, the cupola, irritates the hair cells. It is not known how the membrane of Corti is moved.

From these experiments the essayist drew the following conclusions:

1. If animals are exposed to very loud noises for several hours on many successive days, the entire organ of Corti becomes atrophic and finally is displaced by connective tissue in all convolutions of the cochlea.

2. If animals are exposed in a similar manner after removal of the anvil, atrophy does not take place, showing that hearing is seriously interfered with if the middle ear is not intact.

3. If animals are exposed in a similar manner to clear sounds —i. e., sounds without overtones, the atrophy of the organ of Corti is circumscribed. The area of degeneration is located

nearer to the base of the cochlea as the sound which causes the degeneration is pitched higher. The lower in pitch the sound which causes the degeneration, the higher in the cochlea is the area of degeneration.

4. If very loud shots are fired close to the ears of the animals (without any bodily injury except by the noise) the animal becomes unconscious and remains in this state for several hours or even days. If the animal is killed while in that state the hair cells and Deiter's cells are found in complete disorder; some of them appeared as if torn out of their place and laid in different positions, others looked as if depressed or crushed.

5. If animals are examined at different periods after being exposed to the noise of loud shots, the organ of Corti is found in different stages of regeneration. After six weeks the reparation is complete but the animal is fretful, psychically altered (shell shock).

6. If the animal is exposed the second time to the noise of a loud shot close to the ear, the same change is found immediately after this exposure, but subsequently no regeneration takes place; the whole organ of Corti is replaced by connective tissue, the animal is completely deaf and the atrophy progresses to the cochlear nerve, the spiral ganglion and the acoustic nerve.

7. In birds the organ of Corti is much wider and flat, not rolled up in the form of a snail, as is the case in higher animals, but similar results were obtained. After the shooting the following facts were observed: The outer parts of the organ of Corti were much more changed than the inner parts. The outer hair cells were crushed and torn out of their position while the inner hair cells were practically intact. This led to the final conclusion that in the act of hearing Corti's membrane does the vibrating and not the basilar membrane. In the shooting experiments the end of the membrane makes the largest excursions and does the greatest damage; near the insertion the excursions are smaller and consequently the inner hair cells remain intact.

DISCUSSION.

Dr. George E. Shambaugh expressed himself as quite in accord with Dr. Holinger that this research was one of the

most interesting as well as the most instructive investigations that has yet been undertaken on the internal ear.

When Wittmaack first published the results of his work Dr. Shambaugh examined the drawings with a great deal of interest, but could not help being somewhat skeptical about interpreting slight changes as being the result of overstimulations. He has so often observed in his own work changes in the organ of Corti which were artefacts. Fixing fluids penetrate the labyrinth very poorly and the delicately constructed organ of Corti soon undergoes postmortem changes.

Dr. Shambaugh has often resorted to the extraction of the stapes in order to permit the more rapid penetration of the fixing fluid. When this is done without tearing the endosteum covering the footplate, a very good result can be obtained. To obtain fixation, however, by the ordinary method of dropping the tissue in the fixing fluid does not always give satisfactory results. Often the organ of Corti in the basal coil was in perfect condition, but postmortem alterations were prevalent throughout the upper coils.

An opportunity came some few years later to examine the preparations made in Siebenmann's laboratory, and there was no room for confusing the actual degenerations with any artefacts. The particular things of interest brought out in this research are that different areas of the organ of Corti undergo degeneration with the different types of whistles used in the stimulation. The higher the pitch the lower down toward the beginning of the basal coil was the degeneration area. It was noted that for each tone used a rather extensive area of the organ of Corti was involved. It would seem that a single tone produces stimulation of rather an extensive area in the Corti's organ. This does not in any way conflict with our conception of a peripheral tone analysis. All that is required to make this possible is that a different group complex of hair cells are stimulated for each tone in the scale.

It was very interesting to note that where the conduction mechanism was broken by the extraction of the incus, this method of overstimulation did not produce the characteristic degenerations in the cochlea. The results of loud explosions were particularly interesting. Where the organ of Corti was examined immediately after the explosion as, for example, the

shot of a pistol close to the ear, one invariably found Corti's organ as though blown to pieces. If, however, the animal was not killed until some weeks later, one of two things was found. Either the organ of Corti had undergone complete degeneration or there had been a restitution of the normal condition. This was exactly what was found so frequently during the war. Some of these cases remain permanently deaf, and others show a partial or even a complete return of hearing later on.

Some of the men who were studying these cases during the first period of the war tried to explain these phenomena on the basis of an injury to the nerve fibers in the brain. Dr. J. Gordon Wilson seemed to be inclined to this view. He thought it was Dr. Holinger who, when Wilson made his first report here, called his attention to this experimental research by Wittmaack and Siebenmann. Later Dr. Wilson accepted the view that the defects in the hearing caused by concussion were the results of peripheral alterations.

Dr. Shambaugh stated that the society should feel grateful to Dr. Holinger for bringing this very interesting study before them.

Dr. Joseph C. Beck thanked Dr. Holinger for bringing out this work and called attention to the work done by Yoshi, who is really the great man in Tokyo today. He did a great part of this work and never for a moment forgot or neglected to point to Siebenmann as the man who originated the work. He was kind enough to show Dr. Beck how to preserve a specimen from a case of otosclerosis, in the event he got a postmortem where he could get the labyrinth and fix it, and he showed him how to inject the labyrinth. Dr. Beck had found, as others had, the great amount of artefacts when the specimens were removed by the old methods of fixation. Yoshi took an animal (guinea pig) and thrust a trocar into the heart and then by gravity injected the entire head, including the labyrinth. He then removed the temporal bone and showed him how wonderfully the labyrinths were bathed in the injected fluid. Dr. Beck thought this work of Yoshi's was a very valuable contribution.

Dr. Robert Sonnenschein thought that two of the outstanding points in the address were, first, the light thrown

upon the function of the tectorial membrane to which, as had been stated, Dr. Shambaugh called attention years ago and was the first to offer a better explanation for the theory of hearing than was given by Helmholtz, who used the basilar membrane as the resonating membrane. Second, was the conduction of sounds by the middle ear and the absence of sounds when this conducting mechanism was eliminated.

Dr. Sonnenschein asked if it would be feasible to convey sound by bone conduction alone by the fixation of an apparatus of some sort to the head of an animal after destroying the middle ear. It was assumed by Bezold that the only difference between air and bone conduction is that in air conduction the waves strike the flat surface of the tympanic membrane, whereas in bone conduction the sound waves impinge upon the edge thereof.

Dr. J. Holinger (closing the discussion) said that experiments had been made with two different whistles, one of lower and one of higher pitch, at the same time. In those experiments two areas of degeneration had been found separated by a normal part of the organ of Corti.

The fixation of the labyrinth was carried out by injecting the fixing fluid into the vascular system of the living animal, thus killing the animal. This was the quickest and best fixation that could be obtained and explained why the specimens were so complete.

The controversy between Siebenmann and Yoshi on one and Wittmaack on the other side had lasted for many years. Wittmaack attributed the changes in the cochlea to bone conduction. He argued that the animal was sitting on a sheet of metal, and the sound went through the bones of the body to the labyrinth. As a matter of fact, he exhausted the animals by continuously shaking them, day and night, plus the excessive sound. Siebenmann exposed his animals only eight or twelve hours a day, a period similar to the working hours of boilermakers and engineers. To prove that air conduction and not bone conduction produced the changes, the animals were placed on cotton or some other sound absorbing substance, so that no sound could pass from the cage to the animals' bones. In this way, by air conduction exclusively, he produced the same changes.

This is of great importance for the prevention of locomotive engineers' and boilermakers' deafness: If the floor upon which these men stand was responsible for the transmission of sound and therefore for the deafness all that would be necessary would be to put a nonsound conducting material on the floor for the man to stand on. This was tried and proved of no avail. Wittmaack had furthermore suggested that soldiers during artillery fire should either stand on their tiptoes, or on soft soil, but this too had no effect.

It soon became evident that the Japanese with their nimble fingers were the proper technicians for this work. The handling of the extremely delicate specimens, especially of birds, was very difficult. But for the mapping out and planning of the work, for criticising the experiments, passing judgment on the specimens and the composition of the text, Siebenmann's thought, work and even style can easily be recognized everywhere in the works published under the names of Yoshi and Satah.

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Meeting of Monday Evening, February 5, 1923.*

THE PRESIDENT, DR. C. H. LONG, IN THE CHAIR.

DR. SAMUEL SALINGER presented a boy afflicted with a

**Vitreous Abscess Following Nasal Sinus Thrombosis.**

The case was presented because it was an unusual complication. The sinus phlebitis developed about two weeks after the onset of a suppurative otitis media with fairly typical symptoms, except that the chills were absent. The day before operation he began to develop pains in the ankles and other joints. At operation they found a mastoid abscess, a large extradural abscess and a lateral sinus thrombosis with pus within the lumen of the vein. The abscess was cleaned out and the internal jugular ligated. (Cultures from the mastoid as well as the thrombus showed chiefly staphylococci.) Four days later the boy developed a swelling of the right lids, both becoming very edematous and the conjunctiva greatly injected. The humors were clouded so that the fundus could not be seen. It was thought that the boy was developing a cavernous sinus thrombosis, but because there were no symptoms of involvement of the extraocular muscles and in the absence of exophthalmos it seemed that this might be an error. In a few days the swelling diminished, the anterior chamber cleared up and one could distinguish a vitreous abscess. At the time of presentation the eyeball was somewhat shrunken and the yellow accumulation within could be clearly seen through the cornea and unimpaired lens.

Dr. Salinger had never met with a vitreous abscess following a lateral sinus thrombosis. The boy has practically no light perception, and the Doctor has been informed that the sight is permanently lost, but there is no metastatic danger from this source.

DISCUSSION.

DR. HARRY WOODRUFF, Joliet, said that he never had seen an abscess of the vitreous following lateral sinus thrombosis but saw no reason why it could not occur. In any situation where pus might be present in the circulation such an abscess might form. He thought the patient was fortunate not to develop an abscess in both eyes, as occasionally happens following certain infectious diseases, such as scarlet fever. He was surprised that such a case had not been reported before. The fact that the pus cells in the circulation more often locate in the joints and do not affect the eyes he thought showed that they were well protected against this invasion. In his opinion, if the eye atrophied it should be removed.

DR. BURTON HASELTINE was much interested in the case because he saw an apparently identical one ten or twelve years ago. In that case there was a lateral sinus thrombosis, and operation was advised. The patient was seen the following day by Dr. Ballenger, who advised the same operation, although he did not know that Dr. Haseltine had previously seen the case. They both believed that the patient would die, as they thought it was a cavernous thrombosis. The patient did not die, however, and Dr. Haseltine saw her five years later, at which time Dr. Wilder also saw the patient. There had been no operation of any kind, but the patient had been ill for several months and had had numerous metastatic abscesses, some in the buttocks and various places throughout the body. When seen five years later, the eye was completely atrophied, but the patient had made a partial recovery from the arthritic condition.

DR. SALINGER (closing) said that this boy had two other metastases, one on the rim of the left auricle and the other on the scalp in the left occipital region. The operation disclosed an enormous amount of pathology, an extradural abscess, phlebitis extending from the knee to the bulb and a suppurating thrombus within the sinus. He was urged to operate again when the eye condition appeared, but felt he was fortunate in not having followed that advice. The boy was on the eve of recovery and was about to be dismissed from the hospital.

**Paper: "Lymphoid Tissue of the Tonsillar Fossæ Following Tonsillectomy."***

By Samuel J. Pearlman, M. D., and
Benjamin J. Grichter, M. D.

DISCUSSION.

Dr. Joseph C. Beck said he had so many times seen tissue left by the beginner in the use of the ring for the removal of the tonsil that he was skeptical about the development of follicular tissue from the picture of Tilley's work or the work of Poynter in Nebraska showing that there may be recurrences from the plica, as he showed the large lymphoid tissue in the plica which is seen after thorough tonsillectomy. Dr. Beck thought one should be careful about diagnosing a recurrence of tissue after a thorough tonsillectomy. If the essayists had stated that after operation the cavity was thoroughly inspected and those follicles which Tilley's picture showed were there microscopically it might be taken seriously, but if such a well worked up thesis were allowed to go by without some dissension it would give rise to claims of curing tonsil stumps with X-ray therapy. He thought some of those present would recall that at a very large gathering of the American Radiological Society work on tonsil stumps done by Dr. French and others in the East was shown to prove that operations for the removal of tonsils were useless because they recur. Some years ago Dr. Goode read a paper before the society showing that following tonsillectomy there was retrotonsillar tissue. In Dr. Beck's opinion, many of these follicles were the remains of tonsils, whether the operation was done by ring, dissection or any other method.

Dr. Robert Sonnenschein thought a great many would agree that in many cases of complete tonsillectomy, so far as the eye could see, where the tonsil fossa had been inspected by pulling back the anterior pillar and where the fossa seemed as clean and as smooth as possible, weeks or months later one could see small round glistening masses similar to those seen on the posterior wall of the pharynx. In some such cases as those described by the essayists he had seen the fossa immediately after operation, with the pillars retracted, and it was

*See page 860.

as smooth as could be, so to the naked eye there was no sign of anything left. Then with a punch forceps these pieces of tissue were removed and examined microscopically and the conditions described were found.

Dr. L. W. Dean, Iowa City, Iowa, said that he felt that when tonsils were removed because they were known to be a focus of infection, the plica triangularis should be completely removed with the tonsil. In children with diseased tonsils without apparent systemic infection, an attempt should be made to save the plica and get a better cosmetic and functional result so far as the throat is concerned.

Dr. Charles Robertson recalled the case of a little boy, aged 12 years, who came into his clinic with immense tonsils and adenoids. This boy was of such a nature that he did not object to being operated upon and consequently was demonstrated before a class. He performed complete enucleation of the tonsils and complete ablation of the adenoids. In three weeks he returned and presented the appearance of an immense tonsil on either side, with a large adenoid in the nasopharynx which was fully as large as the original one. He was again operated upon and the masses removed as completely as they could be removed, so that one could see the fibers of the constrictor muscles. In about three weeks the patient returned with exactly the same condition. The masses were removed a third time, and following that removal there was no recurrence, but there was a supernumerary tonsil behind each pillar, a beginning tonsil just behind the uvula.

Dr. Robertson thought that in these cases it made little difference how thoroughly and completely the tonsils were removed, as some of the growths recur, and recur in different degrees in different patients. An incision of the skin of one patient will heal without a scar, whereas another patient will have a keloid scar. He had paid particular attention to this question and believed there was a systemic condition which was at fault and which should be investigated. In his opinion, there is a certain condition in which lymphoid tissue and granulation tissue piles up. In the mastoid in one case one will have to burn granulations, while in other cases there will be no granulation tissue to burn, while the wound will need stimulating. One case is exuberant and the other anemic.

He had seen in some cases an exuberant hypertrophy in two or three weeks, which eventually shrunk, leaving a nice smooth cavity.

Dr. Robertson believed that no one could say that the lymphoid tissue developed a crypt, or whether it was pure granulation tissue or a piling up of lymphoid tissue from one spot. He thought much work had to be done on the tonsillar fossa, and no one at present realized the seriousness of the proposition. He had noticed that in particular cases, for instance, in people who were suffering with tuberculosis, if they were operated upon there would be an exuberant granulation tissue pushing up into the tonsillar fossa or some place else. He had seen many such cases. No one had ever studied the matter to determine whether there was anything in the blood that had to do with the piling up of this tissue, and Dr. Robertson thought this offered a field of investigation that would be productive of good results.

Dr. Edwin McGinnis thought this was not a postoperative recurrence of the tonsillar tissue but, as he understood it, pieces of lymphatic tissue which were clipped out of the spaces behind the tonsils. He could confirm these findings in some work that he had done. He did not use the Beck ring, because he felt that it had a tendency to slip, and one might have a fringe of tonsillar tissue left around the wound. In the Sluder operation one often finds the upper part of the tonsil slipping out of the fenestrum and in enucleating the tonsil this is frequently left behind. Sluder has said that occasionally he would buttonhole a part of the tonsil, and it was a question whether there would not be a recurrence from that part of the capsule which was left.

Dr. McGinnis had had some cases which he had been watching to see whether they would develop a tonsillitis and whether the tonsils would regrow. He believed all would agree that in the last few years there had been fewer recurrences because better work was being done. On removing tonsils under local anesthesia, in dissecting back over the top one will frequently find a little lymphoid tissue that slips away from the capsule, and if these pieces are snipped off they prove to be lymphoid tissue. Dr. McGinnis operated on one patient two years ago, doing a very thorough operation, and this patient returned

about two weeks ago with hypertrophy of the lateral mass of the lingual tonsil and a tonsillitis in the hypertrophied part of the tissue.

Dr. George L. Richards, Fall River, Mass., agreed with Dr. Beck that with either the Sluder or any other type of operation it was easy to remove the tonsil and, after passing an instrument all around in the tonsillar fossa, to conclude that one had performed a perfect tonsillectomy. On the other hand, if with a dull retractor one elevated the anterior pillar so as to expose the whole supratonsillar space there would frequently be brought into view a small remnant of tonsillar tissue.

Some years ago, when first doing tonsillectomies, he made a good many microscopic sections and found that the muscle tissue extended into the capsule and that the gross appearance of this tissue on the capsule was by no means an indication, as claimed by some men, of too extensive removal of the tonsil.

Thorough inspection of the entire tonsillar fossa by retraction of the pillars was the only safeguard against leaving behind unrecognized portions of tonsillar tissue.

Dr. Joseph D. Heitger, Louisville, Ky., said that in 1914 Grunwald published an article in the Archiv. für Laryngologie und Rhin., dealing with the tissue Dr. Richards spoke of and apparently in the location from which the essayists said they had removed the tissue. Grunwald described five main types of tonsillar formation dependent upon the relation of the plica transversa to the upper and lower poles of the tonsil. The various plicæ undergo various types of change, which influence very markedly the development of the tonsil. Grunwald attributed the aberrant portion of the tonsil tissue, simply the portion snared off, to the development of these various plicæ, but the percentage of these variations was very much lower than that found by the essayists.

Dr. Elmer L. Kenyon called attention to Dr. Richards' remark concerning muscle tissue being found in the capsule and said it was perhaps not commonly known that lymphatic tissue penetrates and forms islets in the capsule. He wondered whether it might not be found upon careful microscopic study that these islets completely penetrated the capsule, and

thought this might explain why lymphatic tissue was found outside the capsule.

DR. SAMUEL J. PEARLMAN (closing) agreed that there was such a fold of tissue as Dr. Richards had described, and it might be that in some instances some of it had been taken for study. However, they inspected the tonsil and fauces as carefully as possible, and many cases did not present these findings. They took sections along the course of the anterior plica, not always up in the angle but always at least half way up, so that there was never any question of getting tissue around the lingual tonsil. He believed there was sufficient anatomic reason for finding this tissue, even after apparently complete removal. Some of the tissue was microscopic and could not be seen with the naked eye. Occasionally they found very small follicles under the microscope; other tissue was diffuse in character. They stated in the paper that they did not know whether this lymphoid tissue would atrophy or hypertrophy and also stated that anatomically lymphoid tissue is often present following apparently complete removal of the tonsils. There is lymphoid tissue in this region continuous with the tonsil and performing the same function, but in all instances it cannot be removed with the tonsil. He did not think the tissue they examined was granulation tissue, as it had none of the appearance of granulation tissue. They had done the best they could, but anatomically it was impossible to remove tonsils 100 per cent by any method whatever.

**Paper: "Differential Diagnosis of Labyrinthitis Syphilitica Tarda and Neurolabyrinthitis Syphilitica."***

BY GEO. W. MACKENZIE, M. D.,

PHILADELPHIA.

DISCUSSION.

DR. GEORGE E. SHAMBAUGH said that he expressed the sense of the society in saying they were all very grateful to Dr. Mackenzie for coming to Chicago and that they all appreciated very much his clear, lucid discussion of an interesting condition. His presentation was so clear that no one could have difficulty in following him.

*See page 847.

Dr. Shambaugh asked Dr. Mackenzie what he had to say about the use of arsphenamin in luetic cases. Some observations have been made which suggest that this drug may have a very specific effect upon the eighth nerve. He could recall three cases where a patient afflicted with lues had developed labyrinthine symptoms with a more or less profound destruction of the hearing and where the administration of arsphenamin had been followed rather abruptly by a very marked exacerbation of the labyrinthine symptoms. The idea that an improvement follows further use of the drug had not shown itself in these cases.

Dr. J. Gordon Wilson said he had listened with great pleasure to the interesting and instructive paper of Dr. Mackenzie. He thought there was no doubt that theoretically electrical stimulation is better than either caloric or rotation stimulation. His experience was that in man the results of electrical stimulation were divergent and not sufficiently sharp and reliable. He used three to four milliamperes and so avoided discomfort and pain. Within the last month he had occasion to observe in man in erect posture electrical stimulation of the vestibular mechanism with such a current and noted that while typical falling reaction was prominent and some vertigo was present there was very little or no nystagmus.

In regard to the hereditary cases, it is recognized that the majority of them become apparent between the eleventh and seventeenth year, usually associated with syphilitic stigmata in other parts of the body. Those are the cases which present the anomalies in regard to hearing and pressure symptoms. In the case of eighth nerve lesions in acquired cases of syphilis, one meets with three varieties: First, where both functions are affected; second, those in which disease falls chiefly on the acoustic apparatus; and third, those which involve chiefly the vestibular. The latter are frequently delayed in their onset, coming first into notice with a crisis.

In regard to treatment, Dr. Wilson had seen, as many others have, acute exacerbations after the use of hypodermic doses of salvarsan, so he now prefers to use mercurial treatment before using salvarsan. In hereditary cases treatment is unsatisfactory and in marked contrast to the results obtained in acquired cases.

DR. ALFRED LEWY said that some years ago he undertook to follow up some of Dr. Mackenzie's investigations of the auditory nerve with the galvanic current. He thought he had learned the proper technic, but found that the attempt became so painful to the patient before a diagnostic conlusion could be arrived at that the test had to be discontinued. He had given up the attempt because the patients did not tolerate it. He thought the tests probably necessitated a special type of apparatus in which the current strength could be absolutely controlled, certainly better than the usual rheostats do it, and a milliamperemeter of greater accuracy and delicacy than the ordinary. The instrument which he used was attached to the street current, and this constituted another difficulty, as the current, at least in Chicago, varies suddenly according to the demands on it. His apparatus was not capable of controlling it to the fine degree of precision necessary to make this delicate test.

Dr. Lewy asked Dr. Mackenzie, in closing, to describe in detail the type of apparatus he used and to state whether or not he used the street current or some type of battery that could be controlled more accurately.

DR. L. W. DEAN, Iowa City, Iowa, said he had been interested in the galvanic tests of Dr. Mackenzie for many years. He asked Dr. Mackenzie if the late manifestations of syphilis occurring in tabes were shown by his tests to be labyrinthitis or neurolabyrinthitis.

DR. JOSEPH D. HEITGER, Louisville, Ky., said that his experience was similar to that of Dr. Wilson. He had talked with Dr. Mackenzie and read his book, which he published in 1910, and had tried out the street current, without results. Finally he went down to Dr. Mackenzie and took up the work with him. Dr. Heitger had been looking for a large nystagmus, such as would be obtained in the use of the caloric or turning test, but it was pointed out that the real nystagmus to look for was just a tiny little jerk. He accordingly had an apparatus constructed under the direction of the electrician and Dr. Mackenzie which had a very delicate milliamperemeter, rheostat and polarity changer, and with this a good deal of the pain mentioned by Dr. Lewy could be eliminated by getting the reaction at just the moment it should be obtained, having

the electrodes, the patient's hands and tragus well soaked with salt solution. The greater resistance that is offered, the more pronounced the pain.

A point which had occurred to Dr. Heitger while listening to the paper was that Dr. R. Foster Moore, in a recent book, made the statement that interstitial keratitis could not be prevented by any form of treatment. This was a rather broad statement and, coming from a man of Dr. Moore's experience in ophthalmology, it must be accepted. It was a question with Dr. Heitger whether any of the tarda forms could be prevented even if detected early. In a series of studies of congenital syphilis in the Department of Pediatrics in the Barnes Hospital of St. Louis, including several hundred cases, it is of considerable note that only three cases of the classical Hutchinson triad were found.

Another point was that probably many of the cases slipped through the examiner's hands because a complete functional examination was not always made. A patient might come in complaining of deafness in one ear only, but careful examination might reveal that both ears were affected, one more than the other. Dr. Heitger thought if both ears were tested thoroughly, both with the functional tests of the cochlea and the other tests as given by Dr. Mackenzie, all would have the same experience that he had had, namely, that it was a matter of learning the technic and controlling it carefully.

Dr. Norval H. Pierce said that Dr. Mackenzie exmplified the qualities of his stock which had caused it to persist so long in the bleakest portion of the world, namely, persistence. He had been advocating for years and years the use of the electric reaction in cases of labyrinthine trouble, but for some reason or other it had not become popular. Dr. Pierce was rather inclined to think that the Viennese school in the early days looked askance at it, and perhaps this had had some effect on its general acceptance. He had had the experience that Dr. Lewy and others probably had had. The pain and uncertainty had been caused by an improper apparatus, but that the test was of great value he was convinced and believed the profession was to blame for not using this valuable means of diagnosis more frequently. Dr. Pierce thought Dr. Mackenzie's valuable paper would bear fruit and that the tests would

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be used more frequently, especially if one could differentiate diseases of the terminal apparatus and of the nerve itself. He did not believe that the profession was absolutely dependent upon this method for this differential diagnosis, for the very therapeutic tests Dr. Mackenzie recommended would locate the syphilitic disease. If under mercury the patient recovered there was no doubt that the disease was meningitis; if not, the disease was in the labyrinth.

Dr. Pierce asked Dr. Mackenzie to tell the society what was the best kind of apparatus and the best method by which this means of diagnosis was carried out.

Dr. Samuel Salinger, in connection with the discussion as to the apparatus, asked if Dr. Mackenzie had ever tried applying the galvanic current direct to the drum membrane. Dr. Salinger had done this by means of a sponge placed directly in contact with the drum membrane and had found it possible to obtain a reaction with a current of not more than one or two milliamperes. He was convinced that if one had the proper milliamperemeter they could obtain the reaction with less pain than was possible by the other method and without having to pass the current through the enormous amount of tissue that is necessary when the electrode is applied to the tragus. He was interested to know whether Dr. Mackenzie had ever attempted the test in this way.

Dr. Joseph C. Beck referred to the treatment in the hereditary tarda cases. In the past year he had observed four cases which were very interesting. One was a meningeal case late in life, in which there was a bilateral deafness, both labyrinths being knocked out, with bilateral facial paralysis. He assumed that this condition extended through the internal auditory meatus. This sort of case was of great importance from the point Dr. Shambaugh had mentioned. The patient was placed on arsphenamin, and Dr. Beck had never seen a patient who responded more readily to the intravenous use of this drug. The condition cleared up and the patient left the city, but three weeks later he received a letter from him stating that he was completely deaf and that the face was immobile again. Dr. Beck thought Dr. Mackenzie would recall the work of Dr. Benario showing that swelling of the nerves in the narrow canal occurred in the early part of the treatment, but if fol-

lowed by more arsphenamin this condition would clear up. This had been the experience of many men, among them Hata, who claims to be the man who discovered salvarsan, and Dr. Beck thought this an important thing to bring out. He had had several cases of hereditary tarda in which the symptoms mentioned by Mackenzie were present. He had tried the galvanic current and the man screamed with pain as soon as the current was turned on, claiming that he felt severe pain at the site of application of the current. Dr. Beck was interested to know what type of apparatus Dr. Mackenzie had used that the patients could stand as much as 20 milliamperes.

In a case of hereditary tarda with marked keratitis they placed the patient on a chair and surrounded her with a rubber cover and burned calomel, allowing the calomel fumes to cover the patient's body. It seemed that this form of mercurialization had a very desirable effect on these deaf patients. Dr. Beck had recently seen a boy of about 20, who was absolutely deaf, who gave no reaction to cold water or turning and who had a severe labyrinthitis. Three months later he had a negative Wassermann reaction. That man's ear improved markedly on some days and then again he would be practically stone deaf. He had no reaction to turning, and when the electric current was applied he complained of severe pain and did not tolerate the test.

Dr. Beck expressed his gratitude to Dr. Mackenzie for his excellent paper.

Dr. Robert Sonnenschein said that the change in the bone conduction in early cases of syphilis had been noted, especially by Oscar Beck and others. It was characteristic that before the hearing changed to any degree the bone conduction was very markedly changed. Dr. Sonnenschein asked Dr. Mackenzie whether in the cases where the ear was markedly impaired the change in bone conduction threw any light on the differentiation between labyrinthitis and neurolabyrinthitis.

Dr. Harry Woodruff, Joliet, Ill., speaking in regard to involvement of the eyes, said he had been rather struck with the infrequency with which one encountered involvement of the labyrinth with interstitial keratitis. Interstitial keratitis from hereditary syphilis was one of the more common eye diseases—in fact, phlyctenular keratitis and interstitial kera-

titis form the most common diseases of the cornea in childhood. It had been mentioned that one seldom finds the Hutchinson triad in these cases and that there was lack of results from any form of treatment, and Dr. Woodruff thought the ophthalmologists would agree with this statement. Some had gone so far as to advocate not treating theses cases at all, but he always treated them energetically with some form of mercury. It was true that during the treatment of these cases the second eye would nearly always become involved, notwithstanding the thoroughness of the treatment. The results in these cases were not so bad, so far as the cornea was concerned. It was seldom that permanent and complete loss of vision occurred. Dr. Woodruff recalled a case which he had had under observation for many years with interstitial keratitis and also involvement of the labyrinth. The cornea cleared up very well and the patient had useful vision, while the labyrinth never recovered, although it had not reached a stage that incapacitated her. She had thorough treatment over a long period with apparently no effect on the labyrinth but a good effect on the cornea. Whether this was simply the result of time or of the mercury he could not say.

Dr. Thomas Faith agreed with Dr. Woodruff regarding the appearance of keratitis in the second eye. He recalled that some years ago it was a common notion not to treat syphilis in interstitial keratitis but to regard the disease as postsyphilitic and improve the general health in every way possible, clearing up the indications of the disease as far as possible.

Dr. Geo. W. Mackenzie (closing) expressed his pleasure with the discussion and thanked the gentlemen who took part in it.

Regarding Dr. Shambaugh's question about exacerbations following the use of arsenical preparations, Dr. Mackenzie recalled that just after the International Congress in Boston where Dr. Beck took part in the discussion of the vaccine treatment of middle ear suppuration, the question arose as to what arsphenamin might do to the optic nerve. At that time Dr. Beck published a paper in which he stated his experience with the Herxheimer reaction and other things, including neurorecurrences. He thought this would answer Dr. Shambaugh's question. Before the day of arsphenamin many other

preparations were used, and the big objection was that the arsenic hit the central nervous system too often and too hard, more particularly the cranial nerves, so that Hata, with Ehrlich, tried to obtain a synthetic arsenic preparation that would not do these damages. Their success was challenged for a while when some odd cases of damage to the second and eighth nerves were reported. But in those cases it was found that upon pushing the arsenic preparations still further and harder, these conditions that appeared to be caused by the arsenic cleared up, showing that the apparently bad results were produced by insufficiency of treatment rather than by the salvarsan itself.

Regarding Dr. Wilson's question about the 20 milliamperes, Dr. Mackenzie said that in the paper he stated that he used 20, or as much as the patient could tolerate. Very few of them could tolerate more than 10 or 12 ma. In a deafmute institution in which he did some work he pushed the milliamperage up to 20 in those cases where the patient was particularly anxious to cooperate, but for practical purposes this strength was not necessary. He thought one did not need to use more than 8, but occasionally he goes as high as 12. He had tried 20 on himself and admitted that it was very painful. Dr. Wilson said that his patients did not complain so much of the pain as of the falling reaction. If the technic was good Dr. Mackenzie was sure that it was possible to get reaction that could be observed with the eye long before the appearance of vertigo, and any patient who had a falling reaction because of the galvanic current already had marked nystagmus, the difficulty being that the operator did not recognize it. No one can look into a patient's eye at a distance and tell through the ordinary palpebral fissure just when the nystagmus begins. The nystagmus obtained with the galvanic test is a mixed rotary and horizontal nystagmus, not one form alone but mixed, because the end organs are being stimulated in all three canals.

The first requisite in making this test is a good light. The operator must get into a favorable location, with a head mirror in position and, standing in front of the patient, throw the light into the patient's eyes obliquely. Next, the head must be tilted very slightly backward with the lids elevated by

the thumb of the examiner, so that the equator of the globe may be observed. A movement of ½ mm. at the corneal limbus is equivalent to 2 mm. at the equator. With the head tilted backward and lid elevated, one can distinguish well what would be a very tiny nystagmus at the corneal limbus. The question has been asked, why not try for a big movement? The same objection applies here as in the case of after-nystagmus. In after-turning nystagmus one will notice a wide excursion nystagmus immediately upon stopping turning, which can be seen at a considerable distance through the normal palpebral fissure, which gradually slows down until it ceases altogether. The most essential thing to do is to time the nystagmus, and if one is not a keen observer he will surely miss the fine movements toward the end of the reaction or, say, after fifteen seconds, when he is apt to record the after-nystagmus as fifteen seconds. The careful examiner should study the movements in the after-turning test until they cease altogether. From the fifteenth second on to the twenty-fourth, the movements will be observed to become finer and finer until it becomes a question whether there is any movement or not. He thought it was well to use a split second stop watch and snap the moment the examiner thinks the movements cease and again when he is dead sure they have. It is just as difficult to time the after-nystagmus as it is to make the galvanic test. If one produces with galvanism a big, coarse nystagmus, the question of the personal equation enters too much. For instance, the observer is left to judge the length and frequency of the excursion when they are equal on the two sides. That is surely poor technic. The proper thing to do is to use the least amount of milliamperage that is required to produce the least amount of nystagmus. Nystagmus which is just as fine as that noted after the sixteenth or seventeenth second in the case of after-turning nystagmus. Otherwise the test would be of no value. The same thing applies to the after-turning nystagmus in that we time the least amount of nystagmus.

Dr. Mackenzie thought he might have left some confusion in the minds of some of those present as to the types of syphilis of the temporal bone. He had described during the last several years the types of syphilis he was best acquainted with

and which were possible of differentiation by the galvanic test. There are other forms which he did not feel prepared to discuss at this time. In Providence a week previously Dr. Mackenzie spoke of some other forms that he hoped to put on a sounder basis than they are at present. Parasyphilis or metasyphilis is not tardy syphilis; they are entirely different conditions. Neurolabyrinthitis is not syphilitica tarda, but the result of syphilis of the meninges that every syphilitic patient manifests more or less during the secondary stage. Syphilis is pathologically a perivascular round cell infiltration. This character of infiltration is found more or less evident in all granulomatous inflammations but not so characteristic as in syphilis. When a patient has syphilitic meningitis of the arachnoid, and more especially of the pia, there is not only the meningitis present but meningoencephalitis. Later there is cicatrization, and nothing scars like syphilis. After several years of contraction of the previously inflamed membrane and cortex, atrophy of the cortex of the brain results; in other words, paresis. The same explanation holds in the case of the spinal cord. In the Lissauer zone, where there is no medullary sheath to the fibers of the posterior roots, we have a place where the resistance is weakest that is most affected, and when the pia shrinks it gives rise to the socalled lightning pains of tabes. If it happens to hit the fifth nerve the patient is liable to have (in the cervical type of tabes) tic douloureaux. Dr. Mackenzie recalled a case in which the Gasserian ganglion had been operated on and removed. The patient subsequently was seen in the Neurologic Clinic and, in spite of the fact that the Gasserian ganglion had been removed, the patient still had pains because the Gasserian ganglion is peripheralward of the roots of the sensory part of the fifth nerve. The same process takes place in the eighth nerve, when we have a primary atrophy, due to the effects of the original neurosyphilis and the subsequent contraction that takes place in the meninges. Retrolabyrinthine primary atrophy of the eighth nerve is comparable with primary atrophy of the optic nerve. Dr. Mackenzie did not care to go into this fully at present, because he is still working on it and will not be prepared to give the most definite findings for some years. This is perhaps the condition that Dr. Dean had in mind. In these cases the deafness is due

to primary atrophy of the nerve and not to the Hutchinson's syphilitic triad. Primary atrophy is due to the late effects of earlier neurosyphilis.

Regarding Dr. Heitger's statement concerning the fact that they found only three cases of the Hutchinson triad in 700 cases of congenital syphilis studied in St. Louis, Dr. Mackenzie said that the reason for this was that if a child was affected at 13 years of age with a syphilitic keratitis, that child was usually sent to the eye clinic. If it happened to be affected with difficulty of hearing it was sent to the ear clinic. For this reason very few cases of Hutchinson's triad were found in the syphilitic clinic because the parents probably knew little of the original cause of the eye or ear trouble. Dr. Mackenzie thanked Dr. Heitger for making clear his early experiences, and then his later experience with the galvantic test. He considered Dr. Heitger a real scientist who, when he knew a person had something worth while, made a trip to that person's clinic and stayed there until he had learned all he could of the other fellow's technic. He thought Dr. Heitger would bear him out that he had found the nystagmus present before the patient complained of vertigo and without any falling reaction. If the patient was asked if the test was painful, he would answer "no." Some children are a little fearful if one starts with electricity, but after obtaining their confidence the test can be made quite readily. He invariably makes the galvanic test, and where it is necessary makes all three, and then asks the patient which was the most painful. They invariably say the caloric test is the most distressing, no doubt because it produces, with the head erect, rotary nysgtamus; besides the caloric test is more unpleasant than the galvanic test, for the reason that it is less under control and it is prone to produce vomiting and severe vertigo that is hardly possible with the galvanic. He believed the least unpleasant test of all was the galvanic.

As to the technic, the observation of the nystagmus was of the very greatest importance. Dr. Mackenzie uses an electric apparatus made by Geiger of Philadelphia, which was designed for his particular purpose. He formerly used the current from the Professional Building, in which his office was located, which generated its own current and which was sup-

posed to be 110 volts but really averaged about 90 volts. He has an ammeter which is full jeweled, the same as a full jeweled watch. As to voltage and milliamperage he did not know the exact difference, whether 220 or 110 volts would produce any difference in effect so long as the milliamperage remained the same. Where he is located at present he uses the street current of 110 volts. So far as the ammeter readings were concerned, he could see no difference in the reaction between the current formerly used which tested out at 90 volts and that which he is using at present, which is 110 volts. He was not expert enough to answer the questions raised by Dr. Richards. In his opinion the apparatus could be used with the street current because he is now getting just as satisfactory results as when he used the 90 volts.

A very essential point in the work is to see that the electrodes are perfectly wet. He uses normal saline solution; why, he does not know, except that he had been told by electrical experts and neurologic experts that they used salt solution, and he had adopted it empirically. He has the electrodes dripping wet, with a small ball electrode for the ear with contact that can be made and broken at will. He has a polarity switch in connection with the instrument. It is necessary to have an assistant to control the current and read the meter. He does not know the polarity while making the test, but the moment he observes the nystagmus he notes the polarity. He starts with a low current and is never satisfied with one test. If a questionable reaction is observed at 3 or 3½ m. a. and at 4 or 5, the movements are unmistakable, we know the reaction is positive somewhere between 3½ and 4½ m. a., when we generally credit it as 4. He is not satisfied with one test alone, but repeats the test. It is absolutely necessary to have a good light, to look for eye movements at the equator of the globe, to use a very small ball electrode, or put the electrode into the canal, ignoring the tragus, and to have the electrodes very wet.

As to the electrode applied to the tympanic membrane, Dr. Mackenzie saw no objection to this and hoped that Dr. Salinger would continue using it in that way and give the results of his findings at a later date.

Dr. Mackenzie had obtained some very definite results with 4 m. a. of current. It is known that if a human being is hit in the head with a brick or shovel or the skull is fractured from any cause whereby the petrous bone is not only broken but the fragments are displaced and overriding and the patient bleeds from the ear, not only blood but spinal fluid, then the inner ear must be out of function. In cases of this kind we find mixed horizontal and rotary nystagmus, to the opposite side, with complete deafness. Why does such a patient have nystagmus to the other side? The patient has mixed horizontal and rotary nystagmus to the other side because he has lost the tonus from the ampulla of all three semicircular canals, which was present when the ear was intact. The tonus is lost immediately the labyrinth is destroyed and the balance is in favor of the other side. The amount of loss can be measured with galvanism. The minimum amount of anodal current to produce nystagmus to the opposite side comparable with destruction is 4 m. a. This indicates that the tonus loss by the destruction equals 4 m. a. He has verified this, for although the patient's labyrinth is destroyed the nerve stem is intact and reactive, and by applying 4 m. a. of cathode stimulation to the destroyed side the nystagmus to the opposite side is arrested. Applying 4 more m. a. to the destroyed side, making a total of 8 m. a. stimulation, produces a nystagmus toward the side of destruction.

Dr. Mackenzie extended a cordial invitation to anyone who happened to be in Philadelphia to visit him and said he would be glad to go over the galvanic test with them and demonstrate the technic.